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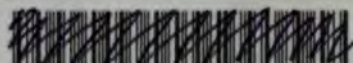
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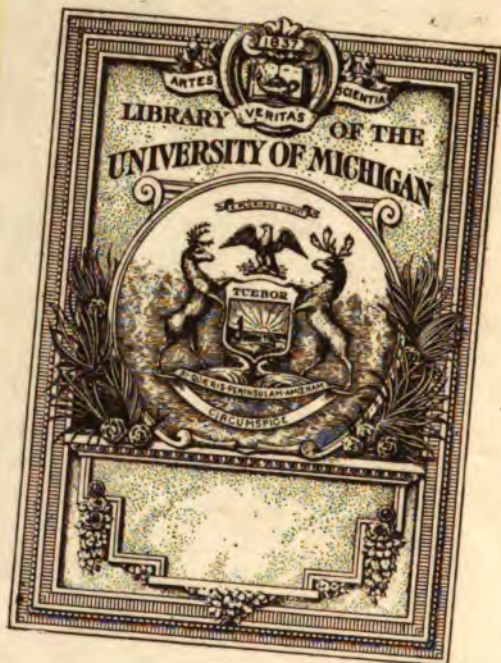
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THE ARCHIVES OF DIAGNOSIS

A QUARTERLY JOURNAL DEVOTED TO THE STUDY
AND THE PROGRESS OF DIAGNOSIS AND PROGNOSIS



FOUNDED AND EDITED BY
HEINRICH STERN
NEW YORK

VOLUME VIII
1915

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HEINRICH STERN, M.D., LL.D.
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Special Articles

PATHOLOGICAL PHYSIOLOGY*

By JOHN B. DEEVER

Professor of the Practice of Surgery, University of Pennsylvania; Surgeon
in Chief, German Hospital,
Philadelphia

Pathology embraces both the structural and functional changes caused by disease. The term pathological physiology is appropriately applied to the pathology of function, but applied as a rule in such manner that the student is prone to dissociate cause and effect; in other words, attention is too often paid to the symptoms of disease to the exclusion of the disorders of physiology that give rise to them. It will not be amiss, therefore, for us to consider some of the broad general principles of the pathological physiology of the digestive viscera contained within the abdominal cavity, with a supplementary review of several phases of this interesting problem upon which recent discoveries have shed additional light.

Clinical symptoms arising from a diseased viscus merely confess its physiological sins.

The fault in function may or may not be dependent upon a gross pathological lesion, but in the event that such lesion does

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exist the disease in the vast majority of instances is a surgical one, demands surgical treatment, and cannot be cured, except in the rarest instances, by other than surgical measures.

What I want particularly to impress upon you is the fact that almost without exception pathological physiology of the abdominal digestive organs denotes the presence of surgical pathology.

You must not take from these remarks my denial that functional disorders of the abdominal viscera occur in the absence of structural changes. Indeed, the foremost problem that confronts the abdominal surgeon is that of clinical differentiation between medical and surgical diseases, for despite every effort it is often-times impossible to determine the cause of a very obscure pathological physiology.

How, then, can we justify the assertion that an alteration in function of an abdominal organ continuing over a long period of time is usually indicative of a surgical lesion? Why, you may ask, will the surgeon assume the responsibility of advising operation to the patient with abdominal symptoms so indefinite that accurate diagnosis is impossible?

It is only by the conviction borne of long experience with the pathological physiology of the living—by readjustment of the clinical picture to conform with the finding at operations frequently advised on mistaken diagnoses and finally by rational deductions from discoveries accidentally made in the search for a cause of persistent symptoms.

In certain conditions, such as duodenal ulcer, we have been enabled in this manner to construct a clinical symptomatology characteristic of the disease. In the case of other inflammatory or neoplastic diseases of the abdominal digestive viscera that are not associated with localizing signs, we have learned that the mere persistence of functional changes usually denote the presence of a surgical lesion.

You are well aware that the normal functional activities of the digestive system demand a harmony of action in the secretory, excretory, absorptive and motor functions that will permit of the digestion of food and excretion of waste products to proceed in the entire absence of any conscious perception on the part of the individual that these extremely complex processes are going on.

Both the chemical and physical reactions in the digestion of food are interdependent and under the control of a communal nerve supply; it is easily conceivable, therefore, that pathological physiology of our digestive organs will cause a dissociation of function among other members of the system. Regardless of the primary seat of the disease, however, and despite, too, its nature, the majority of chronic gastrointestinal disorders express themselves primarily through the medium of gastric symptoms: the stomach is the mouthpiece of the abdominal digestive viscera. It is necessary, therefore, to distinguish between symptom-complexes arising from disease of the stomach itself and those dependent upon diseases of the related abdominal digestive viscera. Medical teaching, as exemplified in the most modern books, implies that the recognition of each deviation from the normal physiology is a simple matter, and the faith of the student in his ability to recognize each condition is shattered only when practical experience has taught him the depths of this delusion. And then, instead of casting aside the false friendship of dogmatic teaching and seeking for the truth at the operation table, he has often become content to label every gastric disorder dyspepsia in one of its fifty-seven varieties.

As a result, prolonged and irrational efforts are made to alter the course of a pathological physiology by means of drugs. This explains the prevalence of inoperable cancer of the digestive tract; of inflammatory lesions that produce crippling adhesions, which, despite surgery's best efforts, condemn the individual to chronic invalidism; this same disregard of the warning note of the living pathological physiology in acute diseases explains the frightful morbidity and mortality of acute appendicitis. It explains also why intestinal obstruction cases are brought to the hospital days after the onset of the condition, and, finally, to this cause must be attributed the chronic inflammatory lesions of the biliary tract, with bacterial invasion of the pancreas by way of its lymphatics, whereby an essential part of this organ is destroyed. Let me repeat, all abdominal symptoms are not indicative of surgical lesions, but I firmly believe that timely operation will only be advised by physicians who are trained to look upon long-continued abdominal symptoms as an indication for surgical exploration. Text-books of the future must be written on the basis of the living pathology re-

vealed at the operating table and not, as in the past, on the terminal pathology, as displayed on the mortuary slab.

The purely functional diseases of the gastrointestinal organs and diseases of these viscera with a minor pathological basis, in other words, medical conditions, are amenable to scientific drug and dietetic measures.

Let it be your practice, therefore, to advise the aseptic scalpel to patients with the history of long-continued indigestion that has failed to improve after one month's trial of proper medicinal treatment. It is your duty to embrace the modernism of pathological physiology, to demand a rational explanation for persistent abdominal symptoms, and to ever remember that disorders of the stomach are the verbal confession of surgical pathology somewhere within the abdominal cavity.

At the outset of my remarks your attention was called to the interdependence of the several functions of the gastrointestinal tract and how any aberration in one function must necessarily influence the others. It is obvious, however, that one or the other of these functions is primarily influenced in each disease, and in the first condition which we will consider, namely, acute intestinal obstruction, the initial change concerns the motor function of the gut tube.

Acute obstruction of the intestine is usually a rapidly fatal disease even in the absence of actual strangulation or gangrene of the bowel, and it is inconceivable that any interference with the motor power of the intestinal musculature could in itself quickly destroy life. Heretofore the early death of these patients has been attributed to shock arising from injury to the splanchnic nerves at the site of obstruction, and to peritonitis. In this conception of the pathological physiology of the ileus, the motor function is primarily deranged with secondary alteration in the protective function of the intestinal mucosa.

As a result of recent experimentation it has been found that peritonitis has little influence in the fatal issue of ileus; in fact, it has been proved that animals will present the classical symptoms of the disease when sterile cultures are obtainable from the peritoneal fluid, the peritoneal coat of the diseased bowel and from the blood. It is evident that the pathological physiology of intestinal obstruction entails a series of changes more complex than mere

interference with the motor and protective functions of the gut walls. The additional factor concerns the secretory activity of the intestinal mucosa, which has been found to secrete a virulent toxin at the site of the obstruction. This toxic product of pathological physiology is capable of producing the typical picture of intestinal obstruction when administered to normal animals. In toxic doses it causes a profound drop in blood pressure, general collapse, lowered temperature and vomiting—in a word, the clinical picture of intestinal obstruction.

The life of experimental animals can be prolonged by injection of a normal saline solution, which fact merely confirms the evidence already given that the fatal factor in the disease concerns the pathological physiology of secretion of the intestinal mucosa, since the saline solution for a time protects the nerve centers against the toxin produced in the diseased bowel.

Bacterial invasion of the peritoneal cavity eventually takes place and undoubtedly adds to the gravity of the condition, but the dangerous initial factor in intestinal obstruction is the toxic product of a pathological physiology. The clinical recognition of this, which is now proved experimentally, has long been made. The late Dr. Price was accustomed to speak of the poisons within the obstructed gut and he advised and practised drainage of the intestine in all cases of obstruction, whether mechanical in nature or due to the paralysis often associated with peritonitis.

The first symptom of interference with motor function of the intestine suggestive of obstruction should create in your minds the picture of a pathological physiology that will soon create a lethal poison, and with the early diagnosis of the condition the necessity of dangerous operation will disappear.

Another abnormal physiological sequence of the abdominal digestive organs begins with a primary disturbance of the secretory and excretory functions of the liver. You will recall that the normal physiology of the stomach and small intestine presupposes the discharge of normal products of gastric digestion into a normal duodenum at regular intervals. In case the duodenum contains fermented or putrid material, the gastric secretion becomes changed and the discharge of chyme from the stomach is delayed, with the result that putrefaction takes place within the stomach.

The normality of duodenal physiology, in turn, depends in large part upon the antiseptic properties of the bile, so that disturbances of the biliary function become the frequent source of gastric disorders.

The most frequent products of pathological physiology of the liver are gall-stones, the chemical constituents of which are present in normal bile and play an important rôle in intestinal digestion. If, for any reason, the drainage of the bile ducts becomes imperfect, if, in other words, the motor function of the biliary system becomes pathological, the bile salts are deposited in the form of gall-stones. The motor insufficiency under these circumstances is only relative, however, since it is dependent as a rule upon inflammatory swelling of the lining mucosa. The clinical symptomatology of gall-stones arises for the most part from the altered physiology of the stomach, simply because nature has endowed this organ with the power of expression of pathological physiology in any upper abdominal organ. The remote effects of toxemia arising from biliary disease is an alteration in the normal function of the renal and cardio-vascular system. A long-continued action of re-absorbed bile and toxins from the infected gall-ducts results in myocardial and renal degeneration with the development of a pathological physiology of these structures that soon becomes irremediable. For this reason alone, and discounting the evil local effects of the disease, patients who present the symptoms of gall-stone disease should be operated upon at once.

And now, just a word concerning the pathological physiology of cancer of the stomach. This varies not alone in carcinoma of different portions of the stomach, but with the various clinical types of the disease as well. The so-called acute cancers run a very rapid course, and as a rule cause death in three months or less from the time of appearance of the initial symptoms.

Cases of this kind give rise to characteristic symptoms, but are not recognized sufficiently early for effective operative treatment. The latent gastric cancers, especially those that involve areas other than the pylorus, proceed often for many years, and eventually cause death in absence of any prominent symptoms referable to the stomach in the operable stage of the disease.

The carcinomas commonly met with in the stomach like the

preceding variety give rise to no characteristic pathological physiology until the disease is far advanced, so that if you await the onset of obstructive symptoms, hematemesis or the appearance of a palpable mass in the epigastrium, the prognosis will be absolutely hopeless. We do not expect a marked deviation from the normal physiology of the stomach when carcinoma first attacks its walls, since the constituent cells of the tumor are structurally identical with the normal gastric epithelium, and, while they serve no normal purpose, these abnormal cells do not alter the gastric physiology in the beginning of the disease.

The first evidence of cancer of the stomach is an indefinite fullness or weight in the epigastrium after meals with loss of appetite, a gradual loss in weight and strength that is usually progressive in an individual who had previously enjoyed perfect health. What is the pathological physiology underlying these symptoms? It is primarily a motor insufficiency whereby the egress of food from the stomach is retarded, with the result that fermentation occurs and the clinical picture of chronic gastritis is produced. With continued progress of the disease, the motor disturbance increases, and promptly a change in the normal physiology of gastric secretion adds to the digestive difficulty.

The very first sign of disturbance with the motor power of the stomach in an adult who has previously been free of gastric symptoms is an indication for surgical exploration of the upper abdomen, for if you procrastinate until the typical pathological physiology presents itself, the malignant cells will have formed irremovable deposits in the adjacent lymph nodes. It is most conservative to be most radical under these circumstances, and if you will learn the lessons taught by the living pathological physiology at the operating table your patients will show few deviations from the normal until the scalpel exposes the offending organ to the light of day.

Another phase of the pathological physiology of the abdominal digestive viscera that is of particular interest concerns the absorptive and protective functions of the large intestine. It has been said that we eat with our small intestine and drink with the large one. The absorption of fluids through the walls of the large bowel is favored by an antiperistaltic action of the musculature of the

proximal segments of the colon whereby the contents of this portion of the intestinal tract are retained until the greater amount of the liquid is taken up by the blood vessels. In inflammatory states of the bowel walls this favors the migration of bacteria, normally present in large numbers in the colon, and the products of localized peritonitis are, therefore, frequently found as the remains of an antecedent inflammation of the large gut.

Localized peritonitis of the upper abdomen other than that arising from gall-bladder disease is found in association with demonstrable disease of the gastric or intestinal walls—usually ulcerative in type, and one seldom hears of congenital membranes, therefore, above the umbilical line. Lane's kink of the ileum, Jackson's membrane surrounding the colon and similar abnormal peritoneal folds are the products of an altered physiology of absorption of the walls of the large gut that at one time permitted of the migration of bacteria normally excluded from the peritoneal cavity.

These membranes which are the remains of an antecedent pathology subsequently interfere with the motor function of the large intestine, and the treatment of the stasis that follows is one of the most difficult problems that confront the surgeon.

The attempt to restore the normal physiology of the large bowel under conditions of stasis have met with slight success. The reasons for surgical failure in this disease have a two-fold basis. In the first plan of treatment, namely, that of excision of the large bowel with anastomosis of the ileum to the sigmoid or similar procedure with the cecum and rectum, the primary operative mortality is so high that the procedure is unwarranted. Any method which does not provide for exclusion of the large intestine fails because the antiperistaltic action of its musculature fills the excluded gut with feces. Not only is the stasis that follows worse than that prior to operation, but the impacted feces predispose the walls of the excluded cul-de-sac to perforation. If anastomosis is made between the ileum and sigmoid with bilateral exclusion of the large gut, the latter becomes a veritable poison factory, and unless an exit be provided for its contents the harmful results of the absorbed toxins soon appear. If any exclusion operation is attempted a mucous fistula should be made, either of the ascending colon or of the splenic flexure of the colon. This step in the

operation of the ileo-sigmoidostomy with bilateral exclusion of the large intestines promises to be the best substitute for the operation of total resection of the large bowel.

You have, no doubt, observed that the failure to find a satisfactory treatment for intestinal stasis has a physiological basis—the normal antiperistaltic action of the large intestine drives the fecal material toward the ileocecal valve, while exclusion of the gut both proximal and distal to the point of anastomosis is followed by abnormalities in secretion and absorption in the excluded gut of the same nature, but in greater degree than simple intestinal stasis.

You have been given merely a glimpse of the pathological physiology of several diseases of the abdominal digestive organs, but enough I sincerely hope to stimulate the desire on the part of each one of you to reduce every symptom of gastrointestinal disease to its actual physiological and pathological cause. By following this Golden Rule of practice the diseases that come under your future observation will not be permitted to go on and on to incurability because the symptoms are incomplete or otherwise fail to conform with a clinical picture that is too often based on the pathological physiology of terminal diseases.

THE DIAGNOSIS OF ABNORMALITIES OF MYOCARDIAL FUNCTION

By T. STUART HART

Assistant Professor of Clinical Medicine, College of Physicians and Surgeons, Columbia University; Visiting Physician,
Presbyterian Hospital

New York

III.

THE EXTRASYSTOLE

In the routine examination of the pulse our attention is frequently attracted by a form of irregularity which has the following characters: the rhythm is for longer or shorter periods that of a normal pulse, but at intervals this rhythm is interrupted by a pause during which one may get the impression that one pulse beat has failed in

its normal sequence; it appears as if one pulse beat had been omitted and the impression is often described as "a dropped beat" or as "an intermittent pulse." When we come to verify our impressions by more careful observation we may find that, during this pause in which we at first thought a beat had been missed, we are able to detect on delicate palpation, a small pulse wave which had at first escaped our attention; this wave is usually much smaller than the waves of the normal rhythm; it occurs at a time which is a little too early for the occurrence of a beat of the normal rhythm and is followed by a pause which is somewhat greater than the interval between the beats of the normal rhythm; this pause is usually followed by a pulse wave which is a little larger and more forcible than the waves of the normal. This irregularity is known as an *extrasystole*. It is evidently the result of a ventricular contraction which has occurred too early and which is less forcible than the normal rhythmic contractions of the heart; it is therefore also known as *premature contraction*. On auscultating such a heart we will detect a rhythmic series of normal sounds interrupted at intervals by a group of sounds which are weaker and occur earlier than those of the normal cycles; this first and second sounds of the weak group are followed by a silence which is considerably longer than the normal diastolic period.

In some of the hearts of this group the extrasystolic contraction will be represented by a single sound only, and no corresponding wave even of an abortive character can be detected in the peripheral arteries. These signs indicate that the premature beat was wanting in force sufficient to open the aortic valve. The question of the opening of the aortic valve depends on three factors: (a) the energy of the premature ventricular contraction; (b) the volume of the blood in the ventricle at the moment; and (c) the blood pressure in the aorta. These factors depend in turn upon the time of the occurrence of the extrasystole. If this comes early in diastole the contractile power of the ventricle will have recovered to only a moderate degree; the volume of blood in the ventricle will then be small and the aortic pressure will be near its highest point; hence it is hardly probable that the aortic valves will be opened and such a premature contraction will be accompanied by the first heart sound only; the second sound, due to the closure of the aortic valve, will

be absent and there will be no corresponding pulse wave. If, however, the extrasystole comes later in the diastolic period, contractility will have more completely recovered; the volume of blood which has passed into the ventricle will be greater and the aortic pressure to be overcome much less; hence the aortic valve will be opened; the second heart sound will be heard and the small extrasystolic wave may be felt at the wrist.

PATHOLOGY AND ETIOLOGY.

In the sections on the physiology of the heart it was pointed out that all portions of the musculature of the heart have the property of excitability, that is that any muscle cell can respond to stimuli at any time except during the "refractory period" which lasts for a short time after the cell has been stimulated. Also that normally stimuli are rhythmically originated at the "sinus node" and sweep over the tissues of the heart in an orderly manner, exciting to activity its chambers in a definite sequence.

If electrical stimuli of the proper strength be applied by means of suitable electrodes to the wall of the heart of the experimental animal (frog, turtle rabbit, dog, etc.), it will respond by a contraction, no matter what portion of the musculature is excited; the activity thus produced will spread downward in the direction taken by physiological stimuli and also from the point of stimulation upward toward the sinus node, i. e. in a direction the reverse of that of physiological stimuli, and the chambers of the heart will contract in the order in which the stimuli reach them. Contractions thus excited from an abnormal focus are known as extrasystoles, and, according to their point of origin, are known as auricular, ventricular, etc.

If, in this manner, the heart is systematically studied by applying stimuli in the various phases of the cardiac cycle while the heart is beating rhythmically, it will be found that for a period beginning just before and extending a short time after systole, the heart is not excitable even by very powerful stimuli, i. e. the heart is in the "refractory phase" because the molecules upon which the fundamental properties of cardiac muscle depend have been decomposed into their constituent ions. Now the extrasystole which has been experimentally produced throws the heart muscle into the "refractory phase"

so that the next physiological stimulus of the rhythmic series arising at the sinus node will reach the muscle cells lower down when they are inexcitable, hence it will be ineffective in producing a systole. The next systole will not occur until it is brought into being by the next spontaneous stimulus which is formed at the sinus node and which occurs exactly at the moment at which it would have occurred had there been no extrasystole. This lengthened diastolic period which follows the extrasystole is known as the "*compensatory pause*." When the time consumed between the last normal heart beat preceding the extrasystole and the normal beat following the compensatory pause is exactly equal to the time occupied by two beats of the normal rhythm, the long diastolic pause following the extrasystole is known as a "*complete compensatory pause*;" when the interval between the last spontaneous systole and the post-compensatory systole is less than the interval between two systoles of the normal rhythm, the compensatory pause is called "*incomplete*."

A study of the compensatory pause in the mammalian heart reveals the following facts: (a) When the sinus node is stimulated the extrasystole is not followed by a compensatory pause. (b) When the auricle is stimulated the compensatory pause is usually incomplete. (c) When the ventricle is stimulated the compensatory pause is complete. These facts may be explained on the following grounds: As soon as the stimulus material at the node is destroyed by its direct stimulation, the construction of the material is immediately recommenced and reaches the explosive point at an interval just equal to the period of the normal rhythm. When the auricle is stimulated early in the diastolic period (see diagram A) the stimulus is conveyed not only to the ventricle but also upward to the node and will destroy the spontaneously forming stimulus material at the node before it has reached the explosive point, hence the interval between the last physiological stimulus and the post-extrasystolic stimulus will be somewhat less than two cycles of the normal rhythm. When the auricular stimulation occurs somewhat later in diastole the retrograde stimulus may reach the node coincident with the explosion of the rhythmically formed stimulus material, hence in this instance the post-extrasystolic pause will be fully compensatory. When the ventricle is stimulated (see diagram C) the retrograde stimulus reaches the sinus node during its refractory

period just after its physiological stimulus and the post-extrasystolic stimulus will exactly equal the period between two beats of the normal rhythm and the post-extrasystolic pause will be fully compensatory. This explanation indicates how extrasystoles arising from different parts of the auricles may have compensatory pauses

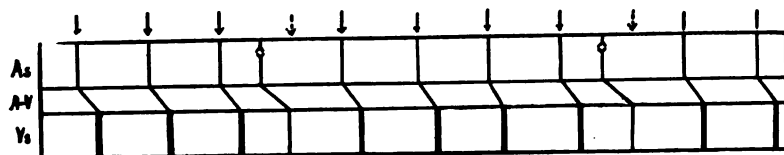


Diagram A. Extrasystole arising from the auricle near the sinus

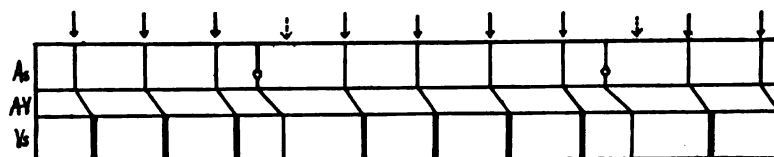


Diagram B. Extrasystole arising from a point low down in the auricle

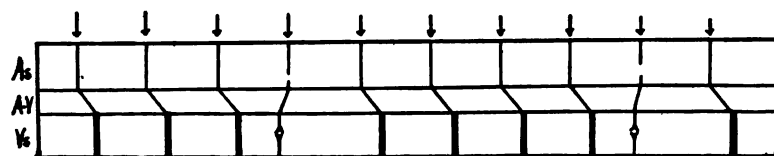


Diagram C. Extrasystole arising from a point in the ventricle.

Diagrams to illustrate the mechanism of the extrasystole starting from various parts of the heart muscle. The arrows indicate the points of origin and the directions taken by the stimuli. Dotted arrows indicate the time at which the normal stimulus at the sinus node should reach maturity if its formation was not interrupted by the extrasystole. The thickness of the lines representing ventricular systole indicate the relative effect of the normal beat and the extrasystole in maintaining an adequate circulation. As = auricular systole. A-V = auriculo-ventricular bundle. Vs = ventricular systole.

either complete or incomplete. It may be stated, as a general rule, that the nearer to the sinus node is the point of stimulation initiating an extrasystole, and the earlier it occurs in diastole, the shorter will be the post-extrasystolic pause; and, conversely, the farther from the sinus node is the point of origin of the extrasystole and the later it occurs the more nearly will the post-extrasystolic pause be compensatory.

Electrocardiographic studies have further shown that the stimuli originating extrasystoles may pass over the musculature of the heart

by the normal paths (nomodrome extrasystole), or, since the stimuli may originate from some point far removed from the normal path or may be shunted from this path by abnormal conditions of the muscles which form an obstruction to their passage, they may take an unusual course through the cardiac tissue (allodrome extrasystoles). A discussion of these abnormal paths and their variegated but characteristic electrocardiographic records will be left for a later paragraph.

Extrasystoles have been produced experimentally in many ways other than the employment of electrical stimuli. Mechanical irritation, heat, the application of irritating salts, obstruction of the great veins (Stassen), clamping of the aorta (Hering), ligation of a branch of the coronary artery (Lewis), the injection of digitalis and atropin (Cushny), adrenalin (Kahn), muscarine and physostigmine (Rothberger and Winterberg). Under proper conditions extrasystoles have been produced in the isolated perfused heart and in the mammalian heart in situ after all nervous connections have been severed, hence it is probable that their cause is an increased excitability of the muscle cells usually quite independent of nervous influences, though Kraus and Nicolai have produced them by vagus irritation.

The conditions of the experimental production of extrasystoles have been set forth at some length since it is upon inferences from these data that our conception of the pathological conditions underlying the extrasystole, as met with in man, is based. Very little indeed is known of the histological changes associated with the production of extrasystoles and there still remains here a field for careful and exhaustive research. Clinically extrasystoles are found far more frequently in those with slow hearts and often they may be made to disappear by moderate exercise which quickens the heart rate. The experimental evidence seems to indicate clearly that the extrasystole occurs because some cardiac muscle cells become more excitable than those of the sinus node and it is therefore on this ground, easy to understand why an increase in excitability should be more apparent during a slow rate, since in the faster rates the excitability of the node is greater than in the slow rates; under such conditions the abnormal irritability of some portion of the auricle or ventricle must be considerable to make itself evident.

It also seems fair to assume from the experimental evidence that nutritional disturbance may play an important part in increasing the excitability of heart muscle; an atheroma with a narrowing of the coronary artery or one of its branches may be the pathological counterpart of the ligation of the branches of the coronary which has been shown by Lewis to regularly produce extrasystoles.

Numerous toxic agents are known to be associated with the production of extrasystoles; they are quite common in many febrile conditions, notably in acute rheumatic fevers. One of the very common phenomena produced by the administration of large doses of digitalis (at least to patients having damaged hearts) is the appearance of ventricular extrasystoles, on the withdrawal of this drug they disappear. Nicotine is another of the cardiac poisons which is clinically prominent as a cause of extrasystoles. The "tobacco heart" is one in which premature beats have become so frequent as to make themselves uncomfortably evident. Excessive tea drinkers are subject to this form of irregularity. Premature beats are found in persons of all ages; they are rare in the first decade of life and are most common after the age of 50. They are considerably more common among men than among women.

Extrasystoles are probably very much more common than is generally supposed; it has been estimated that a majority of persons reaching middle age have had extrasystoles at some period. They are frequently met with in those who afford other signs of impairment of the heart, such as valvular disease, myocardial degeneration and the cardiac complications of nephritis, but premature contractions are also not uncommonly found in those whose hearts have no discoverable abnormality other than this irregularity.

Premature contractions are exceedingly common in individuals of the neurotic type; they may sometimes be induced by irritation of the skin and in persons subject to this irregularity, merely plunging the hands into cold water is sufficient to develop it. They are often associated with digestive disturbances, particularly when accompanied by flatulency. As has been mentioned exercise will frequently cause the temporary disappearance of extrasystoles, but if carried to the point of fatigue the irregularity is prone to become more evident than before. In those predisposed to them, suspension of respiration for a few seconds will sometimes induce these

premature contractions. When present in the upright position they will often disappear as soon as the subject lies down, even though this change in position is accompanied by a slight diminution in the rate of the heart. Extrasystoles are quite common during convalescence from infectious diseases.

IDENTIFICATION

Clinically, the starting point for establishing the presence of the extrasystole is to determine whether the patient has a fundamentally normal cardiac rhythm, which is broken on occasions more or less frequently. When the interruptions occur at infrequent intervals, as is the case in the majority of these patients, the detection of the fundamental rhythm is comparatively easy. If one palpates the radial artery there are long periods during which the pulse is perfectly regular, then occasionally this regular rhythm is broken by a pause which is too long to fit the fundamental rhythm, or one may detect a very small pulse wave followed by a pause longer than that ordinarily separating the waves of the normal rhythm. When one listens to the heart sounds they will be heard for long periods as a normal rhythmic series until this series is broken by the occurrence of one or two indistinct heart sounds which follow the last normal sounds too early and which are in turn followed by a pause longer than that occupied by the interval between the heart sounds of the periods of normal rhythm. The small premature waves detected in the radial and the indistinct premature first (or first and second) sounds heard over the precordium, each followed by a more or less complete compensatory pause, are our usual common evidences of the presence of extrasystoles. Whether one hears at the time of the premature beat a first and second heart sound or only a first heart sound depends, as has been pointed out in a preceding paragraph, on whether the extrasystolic contraction has, or has not opened the aortic and pulmonary valves.

If murmurs are present during the periods of normal rhythm, they are much less distinct in the premature cycle and may be absent. The mitral systolic is the murmur which can most easily be detected in the extrasystolic cycle; the presystolic is more rarely heard; while aortic murmurs are absent or shortened in consonance

with the action of the valve which may fail to open, or open only for a brief period. I have recently seen a case presenting extrasystoles in which no heart sounds could be heard, both first and second sounds being replaced by loud harsh murmurs. At the time of the extrasystole one could hear four murmurs following each other at equally spaced intervals. The first and second of these murmurs were louder and a little longer than the third and fourth; the fourth murmur was followed by a considerable pause which was succeeded by a repetition of the two murmurs which constituted the auscultatory evidence of the ordinary rhythmic activity of the heart.

Another type of rhythm which is easily recognized as due to extrasystoles is the so-called "bigeminus." Here the radial pulse shows a rhythmic series composed of a large wave, a short pause, a small wave and a long pause. This sequence is repeated again and again. The repeated recurrence of two pulse waves followed by a pause has given rise to the very expressive term "coupled rhythm." It consists of a wave of the fundamental rhythm followed by a premature beat and its compensatory pause. This rhythm is one of the common manifestations of toxic doses of digitalis. When an extrasystole occurs every third beat it gives rise to a rhythm that was formerly described as the "pulsus trigeminus."

When extrasystoles occur quite frequently and at very irregular intervals it is sometimes more difficult to assure oneself, by the ordinary physical signs, that the irregularity is due to premature contractions, but careful observation will usually discover a fundamental rhythm, interrupted by beats which occur too early, are followed by a pause and each time they appear give the impression of "coupling."

Inspection of the jugular pulse is frequently an aid in making the diagnosis of an extrasystole. The two venous waves which one ordinarily sees during the fundamental rhythm are often replaced at the time of the premature contraction by a single venous wave larger than the others. This wave is due to the inability of the vein to discharge its contents into the auricle at this moment, since the pressure in the auricle is abnormally high, the ventricle being in systole and the auriculoventricular valves being closed. This is, of course, more in evidence when the origin of the extrasystole is in

the ventricular wall and the auricle and ventricle contract simultaneously.

Whether an extrasystole is auricular or ventricular in origin can only be definitely decided by graphic records and yet the trained observer who has sharpened his powers of differentiation by correlating his physical signs with the evidence of the graphic records, can often, by noting the length of the compensatory pause and the character of the heart sounds of the premature beat, quite correctly assign a particular extrasystole to its proper category.

A graphic record of the radial or of the apex beat is often sufficient evidence to establish the presence of the extrasystole. Such a record (Figures 2, 3 and 4) shows a series of similar waves recurring at equal intervals. This rhythm is more or less frequently interrupted by a small wave which occurs too early to fit into the fundamental rhythm. It is followed by a pause longer than that between two beats of the fundamental rhythm, which in turn is followed by a wave which is usually a little larger than the average wave of the rhythmic series and which is the first of a new series of rhythmic waves. In the case of an extrasystole which originates in the ventricle the post-extrasystolic pause is fully compensatory (see Figures 4 and 5). When the extrasystole has its origin higher up in the cardiac tissues the pause is "incomplete" (Figures 1, 2 and 3), the reason for this has been explained in a preceding paragraph (page 12.)

THE POLYGRAM

Auricular Extrasystoles. The jugular tracing throws additional light on the mechanism (Figures 1 and 2). Figure 1 shows a rhythmic series of waves *a c v*, which is several times (at *x*) interrupted by a similar group which occur too early; it is clear that the auricle contracts too soon and is followed by a sequential contraction of the ventricle.

Another case of auricular extrasystole is shown in Figure 2; here the premature contraction of the auricle occurs earlier in the cycle than was the case in Figure 1, so that the auricular premature wave *a'* is superimposed on the *v* wave of the preceding group; the simultaneous contraction of the ventricle and the auricle causes an unusual temporary stasis in the jugular vein, hence this large wave

(*v a'*), The extrasystole is followed by a compensatory pause which is "incomplete."

The *Nodal Extrasystole* is illustrated (*x* Figure 3). In this instance our conception is that the premature contraction starts at a point in the tissues junctional between auricles and ventricles; from this point the stimulus sweeps upward to the auricle and downward

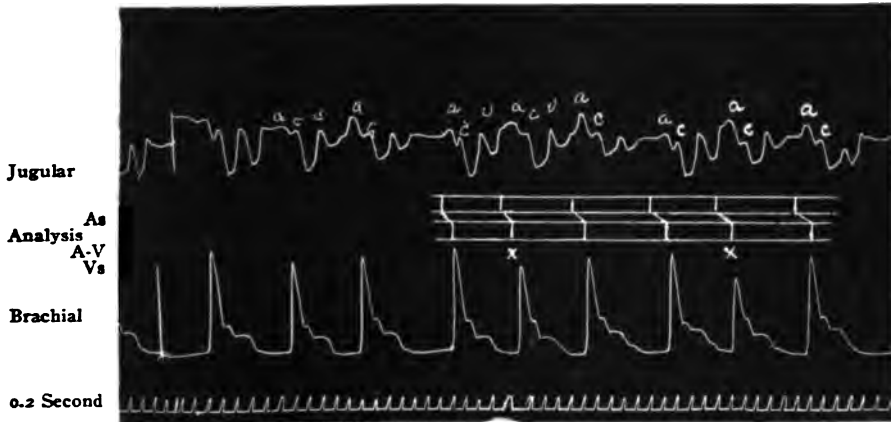


FIG. 1

Auricular extrasystole at x. The compensatory pause is incomplete.

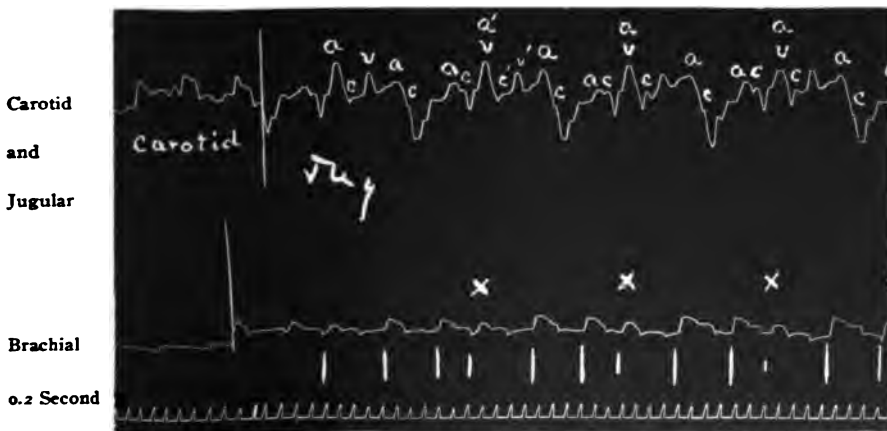


FIG. 2

Auricular extrasystole at x. *a'* of extrasystole superimposed on preceding *v* wave. The compensatory pause is incomplete.

to the ventricle so that these chambers contract practically simultaneously, hence the waves a' and c' of the jugular coincide. The retrograde stimulation of the auricle has destroyed the usual stimulus material accumulating at the normal pacemaker; the building up of stimulus material is, however, at once recommenced and this reaches

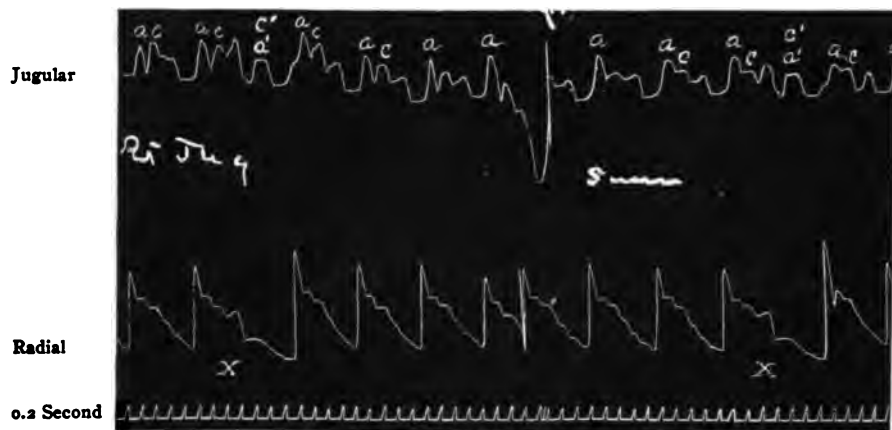


FIG. 3

Nodal extrasystole at x. In the jugular tracing the a and c waves of the extrasystole occur simultaneously. The compensatory pause is incomplete.

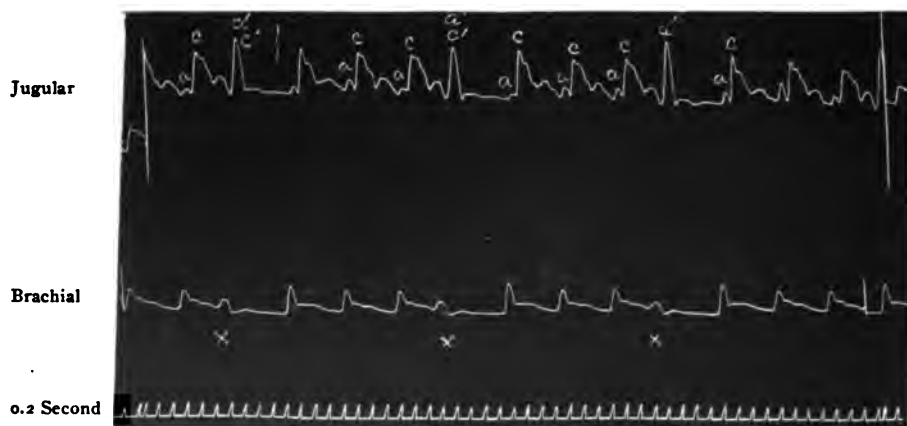


FIG. 4

Ventricular extrasystole at x. In the extrasystolic cycle the auricle and ventricle contract simultaneously (a' c'). The compensatory pause is complete.

maturity in the normal time which is shown by the fact that the time elapsing between the wave a' of the extrasystole and the succeeding a wave is exactly the interval of the normal rhythmic series.

Ventricular Extrasystoles are shown in Figure 4. The auricle, as represented by the a waves of the jugular record, contracts rhythmically, but occasionally (x) the ventricle contracts prematurely so

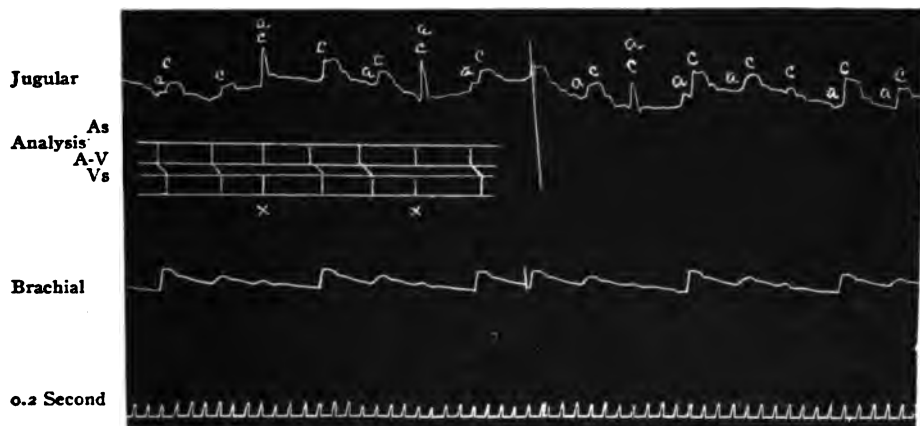


FIG. 5

"Pulsus trigeminus" due to an extrasystole, which occurs every third beat.

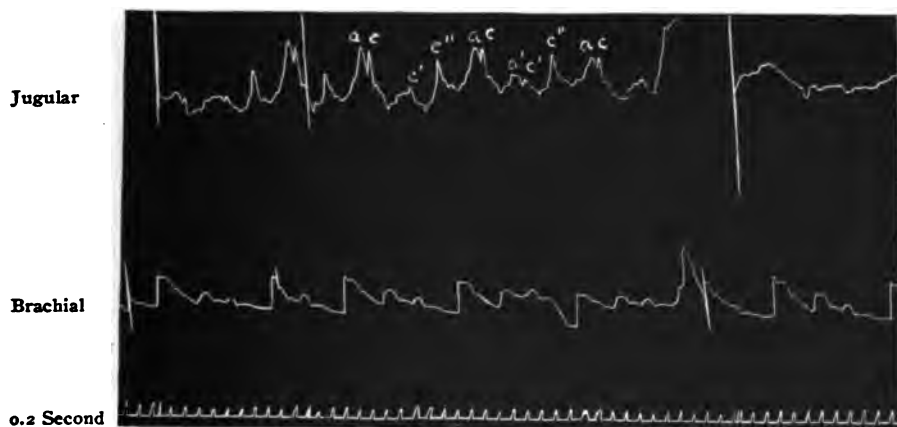


FIG. 6

Ventricular and auricular extrasystoles in a single record. ac = normal cycle, a' c' = auricular extrasystole. c'' = ventricular extrasystole.

that at these times the auricle and ventricle contract simultaneously and their activities are represented by a large wave ($a' c'$) in the jugular tracing. The absence of the v wave in the extrasystolic cycle which is quite evident in the records is due to the empty condition of the ventricle at the time of the premature contraction. It is to be noted that the post-extrasystolic pause is fully compensatory. Figure 5 with its diagrammatic analysis shows a ventricular extrasystole which occurs every third beat giving rise to the so-called "pulsus trigeminus."

Mixed types of extrasystoles are not infrequently seen in a single case. A tracing of such a patient is shown in Figure 6. Here one may make out the following sequence: normal beat, auricular extrasystole, ventricular extrasystole. The analysis of the polygraph in these cases is sometimes quite difficult. The analysis of the tracing shown in Figure 6 was subsequently verified by electrocardiographic records in which the analysis is much less difficult.

THE ELECTROCARDIOGRAMS

As a rule the identification of the kind and point of origin of the extrasystole is most accurately made by means of the electrocardiographic record. The most distinctive features of extrasystoles are that (1) they occur too early, and (2) they are followed by a pause greater than the normal intersystolic pause.

To fix clearly the phenomena which the electrocardiogram discloses, upon which we base conclusions as to the point of origin of the extrasystole, let us recall just what the movements of the string of the galvanometer represent. At any given moment the deflection of the string indicates the algebraic sum of the differences of electrical potential of the heart as a whole. When the stimulus arises at the sinus node (the normal pacemaker) and passes over the heart in a sequential, orderly manner, a series of deflections occur which we have learned to recognize (see Paper 1) as the normal differences of electrical potential for successive instants of the cardiac cycle. If now the stimulus arises from some point of the cardiac musculature other than the "*sinus node*," it is quite evident that the impulse passing by abnormal paths and reaching portions of the cardiac tissues at intervals quite at variance with the normal will produce differences of electrical potential at successive moments of

the cardiac cycle quite different from the normal. How great are the variations in electrical potential which result from the extrasystolic contractions may best be appreciated by a study of the curves which are here reproduced.

Auricular Extrasystoles. When the focus from which the extrasystole arises is at or near the sinus node the electrocardiographic complexes are usually of the normal form. Such a record is shown in Figure 7. It is composed of a series of complexes, each of which

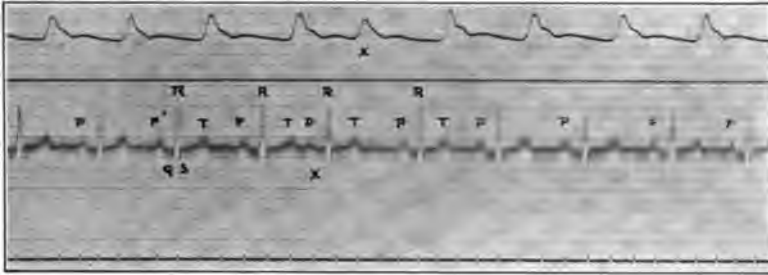


FIG. 7

Auricular extrasystole at x. Compensatory pause incomplete. P = auricular contraction. R T = ventricular contraction. Brachial tracing above.

is practically of the normal type. Each cycle is opened by a *P* wave, which at its proper interval is followed by a normal ventricular complex, *Q R S T*. In the center of the record the fundamental rhythm is broken by a cycle (*r*) which, although normal in other respects, occurs prematurely and is followed by a pause which is not quite long enough to be completely compensatory. This premature contraction must have arisen at or near the sinus node, since the various parts of the cardiac musculature have been stimulated by paths and in a sequence which is the normal one.

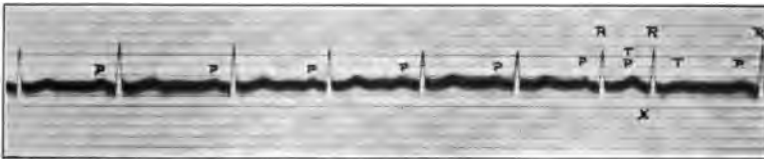


FIG. 8

Auricular extrasystole at x. The auricular wave *P* of this extrasystole is superimposed on the *T* wave of the preceding ventricular complex.

The curve reproduced in Figure 8 shows an extrasystole which has arisen high up in the auricle near the sinus. Here the extrasystole has occurred so early that its *P* wave is superimposed on the *T* wave of the preceding cycle producing a wave which is equal to *P* *T*. The pause following the extrasystole is incomplete.

It has been shown by Lewis* that if the auricle of an animal is made to contract by applying artificial stimuli to various portions of the auricular tissue, the resulting electrocardiographic records will be greatly modified. When the point of stimulation is at or near the sinus node the *P* wave is upward in direction and of a form which we have come to regard as normal; as the point of stimulation is made more and more remote from the sinus the *P* complexes become irregular in form and may be directed downward or show a diphasic variation. We are therefore led to infer that in the human electrocardiogram an upward single *P* wave represents an auricular contraction originating at or near the sinus node; a downward directed *P* wave indicates an origin in the lower part of the auricle; a notched or diphasic *P* wave indicates an intermediate point of auricular origin.

An extrasystole which arose in the lower part of the auricular tissue is shown in Figure 9. The complexes of the ordinary rhythm

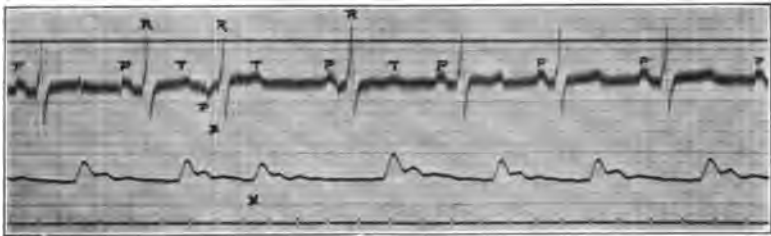


FIG. 9

Extrasystole at *x* arising from a point low down in the auricle. *P* is directed downward in the extrasystole. *P* following extrasystole is diphasic. Below is brachial tracing.

are normal in form except that the *P* waves are rather too broad and have summits which are slightly flattened; the extrasystolic cycle (*x*) is initiated by a *P* wave which is directed downward but is followed by a ventricular complex which is normal in form, indicating that the ventricular response to the premature auricular

*Heart. 1910. II, p. 27.

activity was the result of an impulse which passed down through the *A-V* bundle and over the ventricular musculature by the normal paths in a perfectly orderly manner. It may be noted in passing that the auricular complex which immediately follows the extrasystole has a form somewhat different from the *P* waves of the succeeding normal cycles; this is not an unusual occurrence and suggests that the auricle has not as yet entirely recovered its normal function.

Figure 10 displays a rhythm which was formerly known as the "pulsus trigeminus." It consists of a series of two normal beats fol-

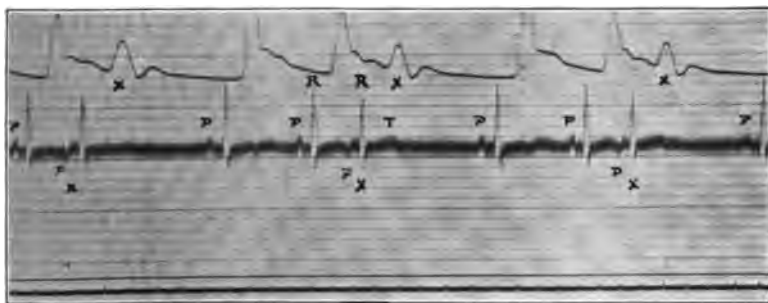


FIG. 10

"Pulsus trigeminus" caused by an auricular extrasystole, which occurs every third beat at x. P is reversed in extrasystole, indicating a point of origin low down in auricle. Radial tracing above.

lowed by an auricular extrasystole. The impression produced on the palpating finger by a pulse of this type is indicated by the radial curve taken simultaneously with the electrocardiogram. All the auricular (*P*) complexes of this record show an unusual diphasic form, suggesting that even those impulses which originated at the sinus node have taken an abnormal path through the auricular tissue. The *P* waves of the extrasystole (*x*) are clearly reversed, indicating an origin low down in the auricle.

The *ventricular extrasystole* presents in the electrocardiogram (Figure 11), a complex far removed from that of the normal ventricular contraction. The abnormal point of origin and the consequent abnormal path which the impulse follows usually produces a much greater difference of electric potential than does the impulse which descends from the auricle and follows the normal path

through the *A-V* bundle and its branches. The auricle contracts at regular intervals, so that often when an extrasystole occurs the ventricular and auricular contractions are simultaneous. The little wave representing auricular activity will then occur during the time

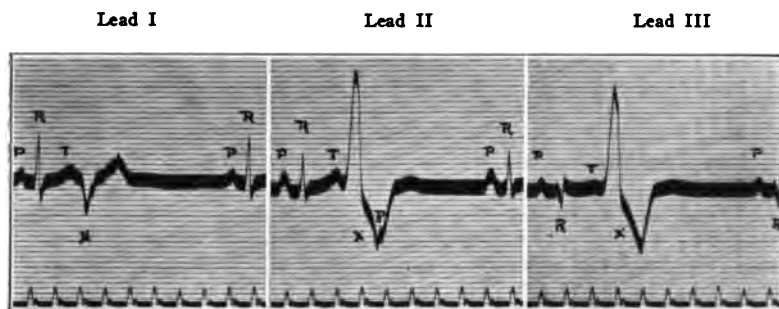


FIG. 11

Ventricular extrasystole at *x* arising from a point at the base of the left ventricle. Showing similarity in the complexes obtained by leads II and III. Compare P R T = normal complex and extrasystolic complex *x*.

of ventricular activity and is usually relatively so small that it is submerged in the large waves of the ventricular complex. Figure 11 shows an electrocardiogram taken from a patient by the customary three leads. The first and last complexes of each lead are the normal for this individual, between these are seen the extrasystoles. It is to be noted that the form of the extrasystolic waves are very similar in leads II and III, but that these differ very materially from the extrasystole pictured in lead I. The similarity of form of the extrasystolic complexes of leads II and III is usual. The complex of lead I may be similar in form to that of lead II, but it is usually quite different. The submerged auricular wave which occurs during the extrasystole can be seen (only in lead II) as a small notch (*P*) in the final dip of the extrasystolic complex.

Systematic studies of the electrical complexes obtained by stimulating various portions of the right and left ventricles both when the branches of the bundle of His are intact and when one of the branches has been cut, have shown that a comparison of the records† taken by lead I and lead II will indicate the point from which the extrasystole has its origin.

†Rothberger and Winterberg. *Archiv. für die ges. Physiologie*, 1913, Vol. CLIV, p. 571.

Lead I

Lead II

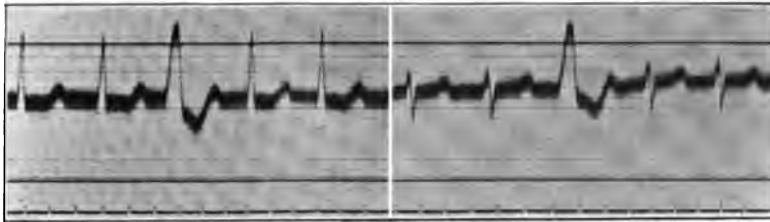


FIG. 12.

Type. 1. Ventricular extrasystole arising from a point in the right ventricle near the base.

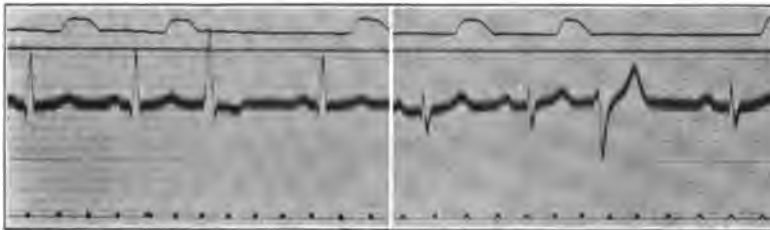


FIG. 13

Type 2. Ventricular extrasystole arising from a point in the right ventricle near the apex. Above brachial tracing the extrasystole produces no arterial wave.

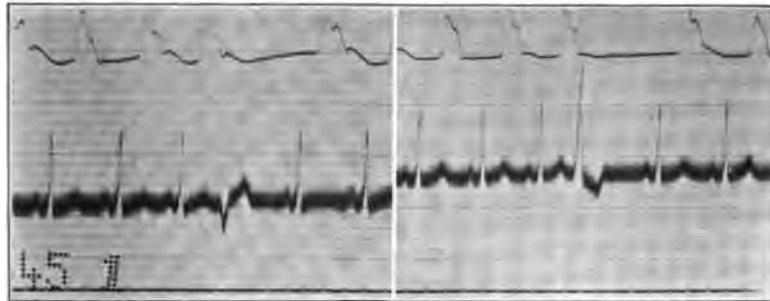


FIG. 14

Type 3. Ventricular extrasystole arising from a point in the wall of the left ventricle near the base. Radial tracing above.

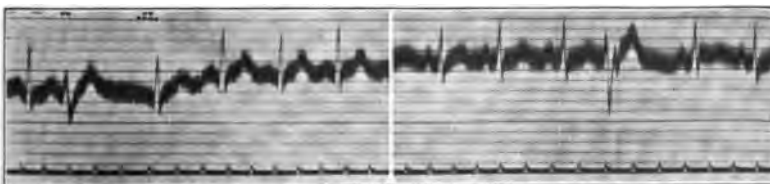


FIG. 15

Type 4. Ventricular extrasystole arising from a point in the wall of the left ventricle near the apex.

The prominent types are shown in Figures 12, 13, 14 and 15. The direction of the principal deflection in leads I and II with the points of origin of the extrasystoles may be tabulated as follows:

TYPE	DIRECTION OF PRINCIPLE DEFLECTION		POINT OF ORIGIN OF STIMULUS.
	LEAD I.	LEAD II.	
1	up	up	Right ventricle near base
2	up	down	" " " apex
3	down	up	Left ventricle near base
4	down	down	" " " apex

A type of curve which is not infrequently met with is shown in Figure 16. Two ventricular extrasystoles appear in this record. Each is preceded by a *P* wave which occurs at its regular rhythmic interval. At first sight one might regard this as an impulse which

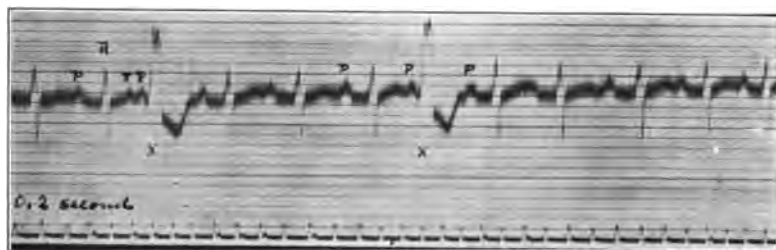


FIG. 16

Ventricular extrasystole at x. The auricle contracts rhythmically, as shown by *P* waves. *P-R* interval = 0.3 second. The extrasystole does not originate in the auricle.

had its origin in the auricle and which was shunted off by an abnormal path through the ventricular wall. One notices, however, that the length of the *P-R* interval of the normal complexes is unusually long (over 0.2 second), while the interval between *P* and the onset of the extrasystolic complex is very brief (0.1 second). It is therefore evident that insufficient time has elapsed between *P* and the onset of the extrasystole to permit of the passage of the stimulus

from the auricle to the ventricle, and we must conclude that the ventricle has contracted in response to a stimulus initiated independently in its own wall.

A contrast to this case is shown in Figure 17. Here the ventricular extrasystole (at x) occurs relatively early and the auricular contraction P is seen as a step on the descending limb of the large extrasystolic wave. The arterial tracing which accompanies this as

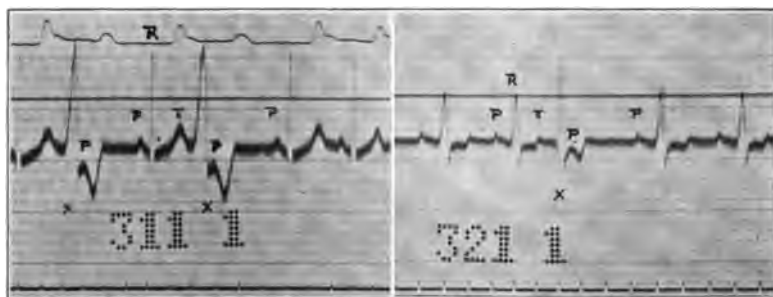


FIG. 17
Ventricular extrasystole at x , showing submerged P waves. Brachial tracing above. Extrasystolic pause is fully compensatory.

FIG. 18
Nodal extrasystole at x . At time of extrasystole auricle and ventricle contract simultaneously. Origin of ventricular impulse is high up in the A-V bundle.

well as many of the preceding electrocardiograms shows the relatively small wave which is produced in the arterial tree by the extrasystole. This evident lack of efficiency of the premature contraction in maintaining an adequate circulation is due to two factors (1) the abnormal sequence of the stimulation of the muscle fibers of the ventricle results in a contraction which is relatively incoordinated, and the propelling power of the ventricles is less than under the normal conditions; (2) on account of the prematurity of its contraction the ventricle is less well filled with blood, hence a smaller volume is expelled into the aorta.

The nodal extrasystole. The majority of extrasystoles which one sees in the clinic have their origin in some portion of the ventricular wall. Auricular premature contractions are far less frequent. A still more rare form of extrasystole is shown in Figure 18. In this curve the extrasystolic complex is only slightly changed from the ventricular complex of the fundamental rhythm, the following pause is fully compensatory and the presence of P in its normal rhythmic

position following the principal wave of the extrasystole shows that the rhythm of the auricle has not been disturbed. Since the ventricular portion of the extrasystolic complex has a form not unlike the ventricular complexes of the sequential rhythm and yet clearly is not the result of auricular activity, we conclude that its point of origin is at some point high up in the auriculo-ventricular bundle and that its subsequent course through the ventricular wall follows the normal channels. This is known as the *nodal extrasystole*.

The *interpolated extrasystole* is another rare form of premature contraction. An extrasystole always ventricular in origin occurs between two beats of the normal rhythm without otherwise disturbing the orderly course of either the auricular or the ventricular rhythm.

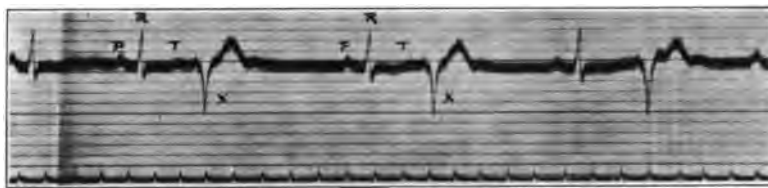


FIG. 19

"Bigeminy." The extrasystoles x(x) arise from the wall of the right ventricle near the apex (Type 2).

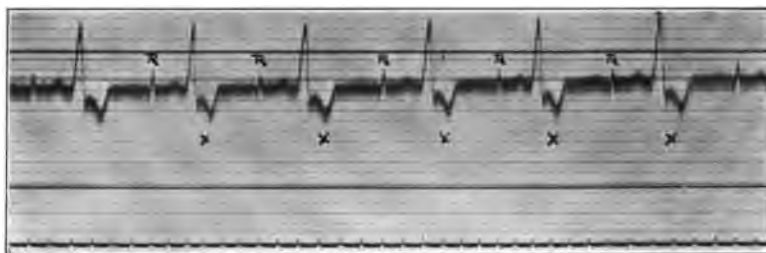


FIG. 20

"Bigeminy." The extrasystoles (x) arise from a point in the wall of the left ventricle near the base (Type 3).

In Figures 19 and 20 are shown two types of "pulsus bigeminus," each due to an alternation of normal cardiac contractions and extrasystoles; the extrasystoles of Figure 19 arise in the wall of the right ventricle near the apex; the premature contractions of Figure 20 arise in a point in the left ventricular tissues near its base.

Extrasystoles of different points of origin frequently are met with in the same patients on separate occasions and sometimes in close succession. Figure 21 shows auricular extrasystoles at A and ventricular extrasystoles at x. The auricular extrasystoles have an in-

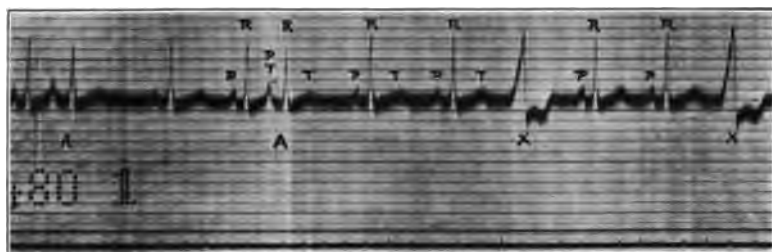


FIG. 21

Extrasystoles from different points of origin. A = auricular extrasystole with incomplete compensatory pause. X = ventricular extrasystoles with complete compensatory pause.

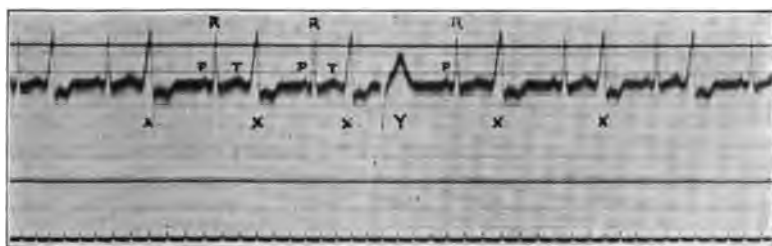


FIG. 22

Two types of ventricular extrasystoles. X arising from the right ventricle near the base. Y arising from the left ventricle near the apex (Types 1 and 4).

complete, the ventricular a complete compensatory pause. Figure 22 shows an alternation of ventricular extrasystoles (x) and normal ventricular complexes. At the center of the record (y) the sequence is further disturbed by the occurrence of a ventricular extrasystole from an entirely new point of origin.

THE CLINICAL SIGNIFICANCE

of the extrasystole is one of considerable importance. Most of us have followed the career of patients who have had occasional extrasystoles for a number of years and often we can secure a history of the existence of this form of irregularity for many years, antedating

our own observations, yet we rarely see a case of cardiac insufficiency which can reasonably be attributed to this irregularity *per se*. The patient is often quite conscious of what they often describe as a "thumping" in the precordial region, "fluttering of the heart," or "palpitation." On examination a large number of these sensations can be shown to be due to the presence of extrasystoles. These sensations are often the occasion of considerable alarm to the patient particularly when they are first discovered and the physician who assures them that this irregularity in itself is of very little significance and rarely is the forerunner of more serious trouble does the patient a great service in removing his grounds for anxiety.

When, however, we see cases which show extrasystoles at very frequent intervals and particularly when the extrasystoles arise from more than one focus our prognosis should be much more guarded, such irregularities are evidences of more serious myocardial defects. The rapid and persistent increase in the number and a multiplication of the foci of origin of extrasystoles point to advancing myocardial changes and are often associated with symptoms indicating cardiac insufficiency. Curiously enough some of the patients in whom I have discovered extrasystoles occurring constantly and in great numbers were quite unconscious of cardiac irregularities.

A more prolonged study of the different types of extrasystoles, their points of origin and their frequency may eventually lead us to modify our prognosis in accordance with such findings, but as yet our facts do not warrant more positive statements. Our prognosis ultimately rests on the extent of myocardial damage, and the extrasystole is merely one of the symptoms which suggest that the defective muscle is little or much affected.

THE DIFFERENTIAL DIAGNOSIS BETWEEN ACRODERMATITIS CHRONICA ATROPHICANS AND DIFFUSE IDIOPATHIC ATROPHY OF THE SKIN

(A CLINICAL STUDY)

By FRED WISE

Instructor in Dermatology and Syphilology, College of Physicians and Surgeons, Columbia University; Chief of the Dermatological Clinic, Beth Israel Hospital; Attending Dermatologist, Montefiore Home

New York

In view of the existing literature on the subject, to publish a paper on acrodermatitis chronica atrophicans may seem a rather fruitless undertaking. It is justified only by the fact that within the last ten years, probably a dozen instances of this rare malady have been encountered and exhibited before the dermatological societies of New York City, and that the questions of diagnosis, classification and nomenclature have frequently given rise to considerable controversy and differences of opinion. The inference should not be made, however, that with this paper the author hopes or expects to put an end to the various contentions of the able and well-informed clinicians, in whose eyes acrodermatitis atrophicans and diffuse idiopathic atrophy of the skin is one and the same clinical entity. Still, an attempt to differentiate the two disease-processes from a purely clinical standpoint, may prove to be not altogether futile.

Under the title acrodermatitis chronica atrophicans, Herxheimer and Hartmann¹, in 1902, first described a series of cases representing a certain clinical type of diffuse cutaneous atrophy. Before this date, the same disease and other dermatoses resembling it, were described under a great variety of titles, such as atrophia cutis idiopathica, érythromélie, erythema paralyticum, etc. Buchwald², in 1883, was the first to publish a clear description of a case of this kind. (For a full dissertation on the cutaneous atrophies, together with a comprehensive index to the literature, the reader is referred to the work of Finger and Oppenheim, "Die Hautatrophien," Vienna, 1910.)

Since the publication of Herxheimer and Hartmann's paper, a great deal of work has been done in connection with the progres-

sive idiopathic atrophies of the skin, more especially by German and Austrian dermatologists. A perusal of the literature gives one the impression that Herxheimer's efforts to demonstrate apparent clinical differences between his acrodermatitis and the ordinary diffuse cutaneous atrophies does not meet with universal approval. In numerous comparatively recent case reports, Herxheimer's designation is either entirely ignored, or the reporter may tentatively offer the suggestion that his case may represent an example of acrodermatitis chronica atrophicans. Often he seems to be at a loss to know under which title his case should be recorded. Even Finger and Oppenheim only grudgingly admit the existence of a clinical sub-variety of diffuse cutaneous atrophy, which Herxheimer and Hartmann individualized by creating for it a new name. In fact, these authors definitely state that they regard acrodermatitis chronica atrophicans as being merely a variant of dermatitis atrophicans maculosa, and not an individual species. Nevertheless, they describe, in a very lucid and highly interesting fashion, two different clinical entities in two separate and distinct chapters; one dealing with dermatitis atrophicans diffusa, the other with acrodermatitis chronica atrophicans. Viewed in a critical light, they really contradict themselves in denying the existence of the latter clinical picture. For if it does not exist, why devote an entire chapter to a dermatosis which obtains only in the minds of its originators? Aside from this apparent inconsistency, the work of Finger and Oppenheim is replete with the most valuable information relating to the cutaneous atrophies, presenting the reader with an enormous amount of original work on the subject, together with a comprehensive and painstaking compilation of the works of other observers. The superiority of their method of classification, together with the excellence of their clinical depictions induces the writer to borrow freely, in the succeeding paragraphs, from this work.

Finger and Oppenheim divide the subject of diffuse idiopathic cutaneous atrophies in the following manner: Under the main heading of *Dermatitis atrophicans chronica progressiva idiopathica*, they include two sub-varieties: (1) *Dermatitis atrophicans diffusa*; (2) *Dermatitis atrophicans maculosa*. *Dermatitis atrophicans diffusa* is again subdivided into two varieties, namely: (1) *Acrodermatitis chronica atrophicans*; (2) *Atrophia cutis idiopathica*.

We are concerned here only with the last two types of cutaneous atrophy; for all other types, including also the atrophic end-stages of diffuse scleroderma, present such radically dissimilar clinical appearances to the eye of the trained dermatologist, that they may safely be eliminated from discussion.

In a previous paper, the writer³ attempted to depict acrodermatitis chronica atrophicans as a clinical entity, possessing a symptom-complex peculiar to itself; and to demonstrate the fact that the symptomatology, clinical appearance, course, evolution and termination of the malady differed, in some respects, from other forms of diffuse atrophy of the skin, therefore entitling it to consideration as a separate clinical entity. In this paper, an attempt will be made to show wherein these differences lie; and to accomplish this purpose, a description of the two forms of cutaneous atrophy is essential.

The dermatoses included under the caption of idiopathic cutaneous atrophy present a peculiar "flaccid" atrophy of the skin, to which Jadassohn gave the name *anétodermie* (*ἀνεγός*=flaccid). Here the normal thickness of the skin is markedly diminished; it appears to be redundant, flaccid, wrinkled and folded, showing a decreased or altogether absent elasticity and is easily raised and pinched between the fingers. Such a condition develops slowly and advances insidiously, without any apparently preceding clinical or histological integumentary changes, without apparent pre-existing morbid alterations of the tissues,—not only of the skin, but of the entire organism; this total lack of etiological data is responsible for the name "idiopathic" cutaneous atrophy.

Of the two main types of the affection, one implicates large areas of the integument or even the entire skin, and is therefore called *dermatitis atrophicans diffusa* or *universalis*; the other involves only small plaques—*dermatitis atrophicans maculosa*. The clinical differentiation of the first type into two subdivisions depends upon the localization of the beginning of the affection on the extremities, or, upon the initial localization, on the trunk; hence the separation of *acrodermatitis atrophicans* (*acro*=extremity) from the other forms of *dermatitis atrophicans*, which may occur on any part of the body. In typical *acrodermatitis atrophicans*, the inflammation, even macroscopically, may be ushered in not only by distinct swelling or edema,

but also by distinct infiltrative lesions. In addition, the morbid process in the latter type is almost always limited to the extremities, involvement of the trunk being rare.

Much as the different types of cutaneous atrophy resemble each other from the clinical point of view, still more do they show their resemblance to each other microscopically. The histopathological alterations in sections derived from cases of *acrodermatitis atrophicans* in its terminal stages, are, generally speaking, identical with the microscopic appearances observed in cases of diffuse idiopathic atrophy and *dermatitis atrophicans maculosa*. Such being the case, the differential diagnosis of the various types rests upon clinical grounds alone; the microscope merely confirms the clinical diagnosis of atrophy of the skin; it gives no hint as to which particular sub-type of atrophy we are dealing with.

ATROPHIA CUTIS DIFFUSA IDIOPATHICA

The disease usually begins by the appearance of plaques showing variations in color from bright red to dark bluish-red. Two types may be differentiated; the plaques may be bright red, rather sharply circumscribed, disappear completely under pressure and are covered with thin, branny scales; or, they may be bluish-red, indistinctly outlined, cyanotic in appearance and without desquamation. In the first type, the appearance is that of a mildly inflammatory erythema, while in the second, it resembles a passive hyperemia. These plaques spread out and become confluent, their coalescence being sometimes preceded by the formation of irregular-network-like stripes and bands; thus larger areas of the skin become involved, while new plaques are forming in the vicinity, or at distant portions of the integument. Shortly after these appearances, signs of *anétodermia* supervene, becoming apparent in the flaccidity of the skin; the thinning and wrinkling usually begin in the central portions of the plaques, without manifesting any marked changes in their original red color. The first change consists of a fine wrinkling of the superficial layers, apparently affecting only the epidermis in the beginning, leaving the appearance seen after the regression of an acute edema. The clinical picture of a fully developed atrophying dermatitis is rather constant and uniform, varying somewhat in respect to its localization. The skin

is dark red, bluish-red and brownish-red; from an intermingling of pigmented and depigmented spots, it may assume a mottled, multi-colored appearance.

The skin is markedly thinned and is so translucent that the underlying veins, tendons and nerve-strands shine through distinctly and appear prominently. Lifting a fold of skin, one has the impression of having a piece of silk between the fingers; large folds of skin may be raised very readily, due to its loose attachment to the underlying tissues; allowing the fold to escape the fingers, the skin very slowly assumes its former position. Wrinkling and creasing of the skin is marked, Pospelow comparing it to wrinkled cigarette paper; others have compared it to the skin of a baked apple.

In extensive cases, the larger folds show a disposition to follow Langer's lines of cleavage. On the back, there is a linear configuration following certain paths, beginning opposite the spine, diverging gradually downward and outward, then arching upward over the lateral portions of the thorax, thence converging over the chest wall with an upward trend. Around the *mammæ*, the folds of skin assume a roughly circular disposition, while over the nates they form flattened segments, extending from the outer and upper to the lower and inner portions of the *glutei*, then bending upward toward the *crena ani*. Around the elbows and knees the folds are arranged in a roughly concentric manner, the arches increasing with the distance from the articulation. Over the extensor surfaces of the wrists and ankles the folds are arranged parallel with the underlying tendons. In localities where the skin is normally loose and easily folded, as on the back of the hands, on the knees and elbows, the wrinkling and fold formation is most prominent; also where there is much subcutaneous fat, as over the buttocks and *mammæ*. The least amount of wrinkling usually takes place over areas where the skin lies near the bone, as over the tibia and ulna, and at the borders of the erythematous plaques which precede the atrophic areas. The surface of the skin is exceedingly dry, free of sweat and fat; the hair is sparse or may be entirely absent. In some areas there is a fine, branny desquamation. The scales may be firmly attached and lend to the skin a mother-of-pearl shimmer. The knees and elbows are areas of predilection for the desquama-

tion. The blood vessels of the skin appear in two shapes. The subpapillary vessels form a fine, bluish-red network, in spots distinctly visible through the translucent integument; such an appearance is, however, somewhat unusual. The subcutaneous veins, especially those on the legs, form prominent, sinuous blue strands of varying thickness, some raised considerably above the surface of the thinned skin; others are not engorged or dilated, but appearing as broad blue bands gleaming beneath the skin. These veins form a network most prominent in the atrophic areas; in the erythematous and infiltrated plaques they are absent.

Such is the clinical appearance of a typical case of atrophia cutis idiopathica. Other cases show certain modifications of this picture. In about a third of the reported cases the dorsal surface of the feet, the anterior aspect of the legs, and sometimes also the forearms, present a condition resembling scleroderma. Instead of the reddish and bluish wrinkled skin commonly seen over these regions, the integument is stiffened, appears to be tense, markedly yellow-white, and can be folded only with difficulty. The borders are usually indistinct; sometimes there is a peripheral zone of wrinkled, reddish-brown skin, fusing with the adjacent normal skin. On the leg this condition is usually seen over the lower third of the tibia, anteriorly, extending to the anterior surface of the ankle and fusing with the normal or atrophic skin over the dorsum of the foot. The skin appears to be tense—an insufficient envelope for its contents—causing a prominence of the underlying tendons which appear as yellow and white strands. The integument is smooth, of a waxy sheen, sometimes speckled with brown pigmented spots. The veins, partly dilated, are prominent and distinctly visible, while the circumference of the leg and foot may be diminished. These appearances are designated by the term “scleroderma-like”; they differ from scleroderma chiefly with respect to the thinning and translucency which is characteristic of them.

These scleroderma-like areas occurring together with cutaneous atrophy have formed the subject of considerable investigation by several authors. The differentiation between atrophy of the skin and the diffuse and circumscribed forms of scleroderma, the possible relations existing between the two, and the incidence of both diseases appearing in the same symptom-complex, have been so

thoroughly dealt with in an article by Rusch⁴, that further comment here would seem superfluous.

ACRODERMATITIS CHRONICA ATROPHICANS

In the publication already referred to³, the writer described in detail a typical example of this malady occurring in a middle-aged woman. He will, therefore, limit himself only to a brief description of the salient points characteristic of the condition.

The disease begins on the back of the hands or feet, or both, in the shape of inflamed patches and edematous, soft, doughy infiltrations. In the great majority of cases the skin of the fingers and toes remain normal throughout the entire course of the disease. The areas of predilection, in the beginning, are the extensor surfaces of the forearms and legs. Itching is moderate or may be absent. The infiltrations are bluish-red in color, reminding one of the nodules of erythema nodosum, without being as sharply circumscribed. These infiltrations must not be confounded with the bluish-red, prominent, hemispherical, hard, sharply circumscribed tumors which appear in the neighborhood of the knees and elbows in the end-stages of acrodermatitis atrophicans; these occur only in association with advanced atrophy of the skin. The primary soft, doughy nodules mentioned above, in the course of weeks and months, gradually assume a bluish and cyanotic appearance, become flattened to the level of the surrounding skin, while the overlying epidermis becomes wrinkled into fine folds. As the infiltration recedes the wrinkling becomes more marked, the area is bluish-red, transparent, thinned, gradually assuming the appearance already described under diffuse cutaneous atrophy. The disease advances upward, toward the groins and shoulders, by means of the peripheral extension of the active border of the process, not by the fusing or coalescence of scattered areas of inflammation or infiltration. In other words, the disease progresses centripetally. On the lower extremity, in the typical cases, the process advances upward to within two or three inches of Poupart's ligament anteriorly, leaving a triangular area on the inner and upper aspect of the thighs free. Posteriorly and on the outer aspect of the thighs, as well as over the buttocks, the process extends upward toward the trunk, coming to a standstill at the crest of the ilium. The anétodermia,

in the advanced cases, is most marked over the knees and buttocks. In the final stages the appearance of the skin is similar to that described under *atrophia cutis idiopathica*.

On the upper extremity the process advances to a short distance above the elbow joint. During the infiltrative or pre-atrophic stage a characteristic phenomenon is the appearance of the so-called "ulnar band." This consists of an infiltrated band of skin overlying the ulnar bone, extending from the wrist to the elbow. In the course of months or even years this strip becomes thinned, wrinkled, atrophic and translucent, its borders gradually merging with the adjacent integument. In some cases the strip is quite sharply margined, so that the contrast between it and the surrounding skin is quite obvious. An analogous strip less often appears over the tibia. The ulnar band appears so consistently in *acrodermatitis chronica atrophicans* that it may be regarded as a characteristic symptom of this type of atrophy.

The above brief description applies to the ordinary types of *acrodermatitis chronica atrophicans*, of which the writer has seen a half-dozen cases in the last ten years. Variations and modifications of this clinical picture are described in the literature. A common sequel to the process is the appearance of the hard, globular tumors near the knees and elbows, mentioned above. According to Finger and Oppenheim⁵, these may also appear in *atrophia cutis idiopathica*, but, according to others, with far less frequency. The scleroderma-like alterations of the integument over the legs and forearms are also common in *acrodermatitis atrophicans*. The translucency of the skin, the *anétodermia*, the chronicity of the process, the absence of subjective symptoms, the maintenance of the general health—are points common to both forms of the disease. The chief points upon which Herxheimer and Hartmann lay stress in their original description of *acrodermatitis atrophicans* are:

1. The occurrence of a primary inflammation and infiltration preceding the atrophic process.
2. The beginning of the disease on the backs of the hands and feet.
3. The slow and insidious centripetal progression, the disease advancing by means of a gradual spreading of the active border.

4. The limitation of the process to certain areas of predilection.
5. The presence of the ulnar (and tibial) band.

A comparison of the two types of atrophy in parallel columns may bring out the differential points more clearly.

<i>Acrodermatitis atrophicans</i>	<i>Atrophia cutis diffusa idiopathica</i>
Atrophy preceded by inflammation, edema, infiltration.	No clinical manifestations of inflammation and infiltration precede atrophy.
Begins on the back of the hands and feet, fingers and toes usually being free.	May begin on any part of the body.
Advances centripetally, by the gradual extension of the active border of the process.	Large areas are usually formed by means of the coalescence of previously scattered foci of the disease.
Areas of predilection are the upper and lower extremities.	No areas of predilection.
Usually an "immune" triangular area below Poupart's ligament.	Not characteristic.
Process usually comes to a standstill opposite the crest of the ilium.	Process usually advances over the trunk.
There is no configuration of skin folds following the lines of cleavage.	Usually seen on buttocks, back and mammæ.
The presence of the ulnar (and tibial) bands.	Usually absent.

In conclusion, it may be said that those observers who still regard the two types of cutaneous atrophy as one and the same clinical entity have good reason to adhere to their opinions, the question being a debatable one. To the writer it seems that the two clinical pictures may well be separated; their separation, however, being justified only by the sum total of their characteristics.

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DIAGNOSTIC VALUE OF HYPERESTHESIA OF THE
SOLAR PLEXUS AND ITS RELATION
TO GASTROPATHS

By MORRIS SCHOTT

Cleveland, Ohio

Like many objective and most subjective symptoms, hyperesthesia over the region of the solar plexus loses a greater part of its diagnostic value if considered from the individual or monopathological point of view, but considering the symptom from the collective or multipathological side it becomes without question of an important diagnostic importance. Just as thermometry in disease is of little value if considered without the other concomitant objective and subjective symptoms in a given case, we may have a parallelism in the importance of our aims in diagnosis in hyperesthesia of the solar plexus.

In the normal state and in the healthy individual the physiological functions of the stomach are performed unconsciously and without sensation, only two conditions being necessary for conscious sensation, namely, hunger and distension of the stomach. In the normal state the stomach is not impressed with tactile sensation, a condition which gives us daily proof in the introduction of the stomach tube—the passing of the tube is not felt after it has passed the cardiac orifice. On the other hand, distension due to fermentation, either by gas, aerophagia or a too large amount of food at one time, will cause a very uncomfortable feeling, and when a certain amount of overdistension obtains it will even cause intense pain.

We see, then, that in the normal healthy individual there exists no consciousness of the stomach.

It is different, however, whenever the equilibrium of the stomach

functions is disturbed either directly or by reflex irritations due to pathological conditions, affecting other abdominal organs and innervated by the sympathetic system.

The semilunar or solar plexus is the largest and richest antastomotic ganglion of the sympathetic system, and pain-impressions are no doubt due to the afferent and efferent sensory fibers having their origin and their terminals in this location.

The physiological function and work of the stomach, like all other normal functions of the visceral organs, being of an unconscious nature, we must admit that all painful functions of the stomach or other viscera must be considered to be of a pathological nature. We are greatly indebted to Albert Mathieu for many of the clearer points relating to localization of the epigastric point, as well as the masterful classification of solar reflex hyperesthesia. Boas has attributed the painful point in question to spasm or contraction of the pylorus, others have claimed that the epigastric painful point was created by pressure by the physician himself and due to suggestibility of the patient, which no doubt is true in cases where the neuropathic element predominates; on the other hand, in the great majority of cases of dyspeptics the hyperesthetic point of the solar plexus can be plainly demonstrated by pressure slightly to the right of the median line and corresponding to the trunk of the celiac axis. Mathieu observed some phthisical patients at the last stage of their disease who were, as is the rule with such patients, also gastropaths. He found the hypersensitive solar plexus point, marked the point with a nitrate of silver pencil, and at the autopsy ran a long needle straight through from the marked point, fixing it securely in the vertebral column and in every instance penetrated the same nervous plexus of the sympathetic. An attempt has been made to measure the degree of hypersensitiveness and Boas has made use of an esthiometer registering the amount of pressure in grams; the instrument, however, is bulky and hardly adapted for general use. Ch. Jean Roux invented a modification of the Boas instrument, much smaller and giving the same results which registers a pressure up to 5 kilograms. Boas claims that a pressure over the solar plexus in the normal state can be carried to equal 10 kilograms before eliciting any sensation of pain, but I would not advise any such

degree of pressure in any case, in fact I think the employment of such force may be a dangerous proceeding in certain conditions.

Personally I use the common baby scale found in almost every physician's obstetrical bag. For its employment I have devised a simple extension on a pressure button with a hook at its extremity on which I apply the scale and am able to register the amount of pressure by traction and reading the amount on the register. It is simple and easily applied.

Considering the amount of hyperesthesia in a given case, there are several factors which may lead us into error. Foremost is the rigidity of the abdominal wall, which especially in some neurotic types of patients is at times very difficult to overcome, and requires the physician's patience until complete relaxation is procured; again we meet with such extreme cases of hyperesthesia that the mere touch—the slightest weight of the bedsheet or merely the atmospheric contact—will cause contraction of the abdominal muscles.

The biliary vesicle either in acute or chronic lithiasis, distension due to obstruction or simple inflammatory reaction, is also hyperesthetic, but the point of greater sensitiveness is located about three fingerbreadths to the right of the solar plexus point. Besides the previous history of the patient will in most instances guide us in our conclusions. On the other hand, biliary vesiculosis is often associated with gastropathic conditions, and we can often draw a clear line between the two painful points which I have called the neutral gastro-hepatic and esthetic point. Registering the degree of sensitiveness has often helped me in the diagnostic differentiation between gastric and cysto-hepatic disease.

We will also have to differentiate a possible epigastric hernia; in order to produce the maximum degree of pain you request your patient to strongly contract his rectus and other abdominal muscles by flexing his trunk from the horizontal position when one can sometimes even feel a small epigastric hernia. By this flexor trunk movement the abdominal muscles become tense and rigid, forming an effective protection of the sympathetic plexus and a previous hyperesthetic solar plexus becomes hypoesthetic by abdominal protection.

In order to gain a better understanding of our cases we should

divide them systematically, and put each case whenever possible into its own class.

1. Temporary hyperesthesia during tardy gastric pains.
2. Permanent neuropathic hyperesthesia.
3. Secondary hyperesthesia.
4. Gastric pains without hyperesthesia of the solar plexus.

(1) Temporary hyperesthesia during tardy gastric pains is a well-defined group frequently met with in our daily practice. The pain usually appears from two to four hours after eating. Sometimes the pain is provoked immediately after the ingestion of a small amount of milk or even water. These patients do not usually suffer when the stomach is empty or while fasting, but the classic characteristic tardy pain appears at a varying period after alimentation. Generally, we may ascribe such tardy pains to an organic lesion of the stomach, associated with hyperchlorhydria. We usually meet with such pains in acute as well as in chronic ulcer of the stomach, in hyperacidity due to other causes or in alcoholic or mechanical gastritis. Occasionally we meet with some cases where the hyperesthesia persists even in the morning or while fasting, but there is always a marked increase in the amount of hyperesthesia of the solar plexus at the actual time of the active and tardy appearance of gastric pain.

(2) Permanent neuropathic hyperesthesia. In neuropaths the relation between the degree of immediate and tardy pain and the solar plexus hyperesthesia reflexes is disturbed. Neuropaths insist that there is, as a rule, no time in which they are free from pain; their hyperesthetic point is shifting from time to time, and the greater sensitiveness has no relation to the time of ingestion. They perceive their pains even in the morning when awakening. Pressure over the solar plexus point will always elicit a pain that is not dependent on any gastric or other abdominal disturbance. Due to moral chaos or excitement, the pain may persist with appreciable variation during several days or even months when a pressure of 200 to 500 grams may cause the most severe pain which no restriction in diet will modify. At no time are these patients free from pain when moderate pressure is applied over the solar plexus point, and we must distinguish the actual pain due to true hyperesthesia from neuropathic, hysterical or moral pain impressions.

(3) Under the term of secondary hyperesthesia we may include such conditions which are caused by conditions other than direct solar plexus sensory reflexes. Secondary solar hyperesthesias are therefore expressions of conditions having their origin at a distant part and respond to sympathetic nerve impulses. Amongst conditions of this nature we may include the different ptoses of the abdominal organs—gastric, hepatic, renal, intestinal (1) reflex irritations of an inflamed appendix, uterine and ovarian inflammatory conditions, etc. Medicinal gastritis (gastrite médicamenteuze of the French authors), a gastritis caused by drugs which irritate the gastric mucous membrane, has hardly a place in this classification, and I mention it only because the etiological factor is often overlooked and neglected. The solar hyperesthesia which is directly due to alcoholism must also be thought of. In my personal experience the hyperesthetic point due to alcoholic gastritis is, however, more towards the central line of the hypogastrium, and, as a rule, follows the line of greater curvature of the stomach.

(4) Gastric pains without hyperesthesia of the solar plexus. Almost invariably when we fail to elicit some degree of hyperesthesia over the point of the solar plexus on pressure in gastropaths suffering with tardy or spasmodic pains, we may conclude that the pathological and etiological factor is located elsewhere. Taking into account even the extreme rigidity of the abdominal walls immediately following a perforation of an ulcer of the stomach or duodenum, we can still differentiate a degree of hyperesthesia of the solar plexus from the surrounding topographical locations, possibly due to extreme irritation of the efferent fibers. Where the absence of hyperesthesia over the solar plexus strikes us most forcibly during the course of the most intensive tardy pains is in the crisis of tabes dorsalis. During the course of the crisis of tabes, even when the patient may suffer the most excruciating pain, a strong pressure over the solar plexus point of 2 or 3 kilograms will show an almost entire anesthesia. No doubt this absence of pressure pain over the point of the solar plexus is due to lesions in the plexus or spinal cord sympathetic fibers, and I believe that we have in the past not insisted sufficiently on this deep-seated anesthesia in cases of gastric spasmodic pains due to tabes dorsalis.

Conclusions. Reviewing what has been said in the preceding, we

must admit that studying solar plexus reflexes, we can draw conclusions which may help us in many cases to clear up some diagnostic difficulties. First of all we may conclude that an hyperesthetic point over the region of the solar plexus in the dormant state of tardy gastric pains suggests a pathological state of the gastric walls, a congestion of the mucosa, hyperacidity or ulcer of the stomach. Neuropathic hyperesthesia, on the other hand, has a widely different aspect, the production of the same not being constant, as is the case in the preceding class, besides, in neuropathic hyperesthesia of the solar plexus we usually detect the concomitant neuropathic elements. The origin of hyperesthesia due to distant pathological conditions can in most cases be traced by a careful and proper study and careful examination of the abdominal and pelvic cavities. Finally the absence of hyperesthesia of the solar plexus during the paroxysm of gastric pains should lead us to consider the presence of a possible *tabes dorsalis*.

There is no question that the examination and production of the solar plexus reflexes should be included in every careful diagnostic investigation, especially since it is a simple method, and in the absence of an esthesiometer can be carried out by simple finger pressure over a point of about one and a half fingerbreadths to the right of the median line and directly over the location of the trunk of the celiac axis.

A PLEA FOR MORE FREQUENT EXAMINATION OF THE LOWER BOWEL IN THE DIAGNOSIS OF DISEASE.

By JACOB GUTMAN

Director, Pathological Laboratory, Jewish Maternity Hospital; Attending Physician, St. Mark's Hospital Clinic
Brooklyn, New York

A plea for a more thorough examination of every organ in the diagnosis of pathological conditions seems unnecessary in these days of scientific medicine, when accuracy is rigorously demanded by the medical profession. However, certain unfortunate occurrences of recent memory impel me to make this very plea. During the past two years my attention has repeatedly been called to a number of cases where due to negligence or even culpable neglect

to examine properly the lower bowel there has resulted the most unfavorable and quite unnecessary consequences. It seems inexcusable that the recto-anal region should receive so little attention. Indeed the organs of this region are neglected not only by general practitioners of medicine, but even by gastroenterologists, whose interest they ought to particularly attract. Let me ask: Has any important study of the physiological or pathological conditions of the lower part of the intestinal tract appeared in print during recent years?

That the rectum and the lower portion of the sigmoid flexure are the seats of numerous disturbances is known even to the general layman. Nor is the subject of proctology at all a new one, for even centuries before the Christian era have rectal diseases been known and pronounced a plague and a curse to suffering humanity. The Bible, for instance, mentions these disorders in more than one place. Why is it then that the rectum is neglected generally in the examination of patients for diagnosis? Is the cause to be ascribed to inefficient instruction upon this subject given in medical colleges? Or are the disturbances of the lower bowel considered too trivial for serious attention? Or is the cause perhaps to be found in the modesty of the patient which interferes with frank investigations? Is it perhaps the unpleasantness of the work which the physician is willing to avoid, or is it a feeling of incompetency that obliges him to shun these parts? Whatever the cause, the rectal disturbances should receive thorough study and examination. No diagnosis is complete unless these organs have been given careful attention. It is a known fact, that carcinoma is a common habitant of this part of the gastrointestinal tract. How often is such a malignant growth in the lower bowel overlooked and allowed to remain undetected until the resultant enlargement of the liver caused by metastases of the cancer necessitates a thorough search for the primary growth? How often are patients having a positive basis in pathological changes of the lower bowel with gastrointestinal disturbances set down as neurasthenics and dyspeptics? We all know how often diarrhea or constipation is ascribed to perfectly innocent causes until an investigation of the lower bowel reveals the actual etiological factor. How many individuals have suffered the loss of comfort and health for long periods from causes located in the sig-

moid and rectum which are not diagnosed? Such conditions are intolerable. There are ways and means, numerous types of apparatus and Laboratory methods affording as critical and exact an examination of the rectum and sigmoid as of any other part of the body. It is possible to diagnose pathological conditions there as easily as elsewhere.

A few illustrations, not the most striking ones, drawn from my own experiences will point out the consequences following the neglect of the lower bowel.

Case I. (Referred by Dr. A. L. Cardozo.) W. S., male, age 63 years, engineer, Scotch. His family or past history presented nothing of interest. His habits—moderate beer drinker (occasionally whiskey), inveterate smoker. As a young man—gonorrheal infection. About 6 months previous to the examination the patient began to feel uncomfortably in the lower part of his abdomen, first more or less in the right iliac fossa, later entirely in the left. To this the patient paid but little attention until the abdominal pain became complicated with frequent bowel discharges. The latter continued in spite of the frequent administration of various astringents by his physician. The diarrheal evacuations soon became more and more frequent, painful and expelled in a gush. Loss of weight, weakness and abdominal distension soon followed.

When referred to me for examination, the patient appeared pale, rather considerably emaciated, with emphysematous lungs, somewhat enlarged heart, sclerotic blood vessels, small liver, distended abdomen tender upon palpation in its lower half. The anal orifice was eroded and sensitive. The stools were very thin, dark colored, fluid, ill-smelling and contained mucus and blood, microscopically and chemically. The proctoscopic examination revealed in the lowermost portion of the rectum several hemorrhoidal knots, some ulcerated, others indurated, the mucosa appeared thickened, succulent, congested and glistening. At about 30 cm. from the anus we came across an encroachment upon the lumen of the bowel projecting from the right wall of the gut. This mass was fixed and of such dimension as to occlude the passage of the intestine and to make the further introduction of the instrument beyond the obstruction impossible. The mucous membrane covering the mass was ulcerated and spread over with discharge. The diagnosis of a

malignant tumor was made, and the case referred to Dr. Bogart for advice as to the advisability of surgical treatment; but the case was beyond surgical relief, as the disease had too far advanced and the patient expired several weeks afterwards. This fatal termination of the case might to a certain extent be ascribed to the delay of an exact diagnosis caused by the tardiness of the patient to consult his physician. An early proctoscopic examination would have revealed the true condition, and proper surgical interference would have changed the entire aspect of the case.

Case II. (Referred by Dr. J. Wheeler Smith.) M. C. McG., 52 years old, female, American. Family history as to occurrence of neoplasms—negative. Married 22 years, 1 child, 1 miscarriage; menopause at the age of 36. For the last 10 years patient was inclined toward constipation with occasional attacks of indigestion, vertigo, irritability and nervousness. There never was any elevation of temperature. The stools were generally hard, consisting of small scybalous masses and of meager volume; never bloody, mucous or diarrheal. During the last few months the constipation became aggravated and the movements of the bowels were preceded by pain of a crampy character felt in the lower abdominal fossa. The physical examination revealed a poorly nourished individual without particularly important pathological changes in the lungs, heart or abdominal organs. Only the left lower abdomen seemed tender to pressure, and an enteroptosis was more or less evident. The administration of a bismuth enema disclosed considerable information. The course of the fluid as seen with the fluoroscopic apparatus was observed to fill the rectal ampulla, which when distended was shown to be of normal dimensions; it also filled the sigmoid flexure without difficulty, but when the fluid attempted to enter the adjoining portion of the descending colon there was a decided interruption in the flow of the bismuth which continued until the pressure under which the fluid was forced into the bowel was considerably augmented by elevation of the irrigating vessel and thus the resistance was overcome. The part of the bowel immediately above the obstruction when filled was shown to be of larger diameter than the rest of the bowels. Otherwise there were no other difficulties encountered in the administration of the bismuth enema until the whole length of the large bowels to the ileo-cecal valve was

filled. Because of the patient's age, the positive presence of an obstruction of the bowels, the site of the constriction, the gradual aggravation of the obstipation, the painful peristalsis accompanying fecal expulsion, the run-down and more or less emaciated condition of the patient—all these symptoms seem to have justified the assumption of malignancy as the cause of the obstruction. The latter condition was already established from the X-ray findings of the case, but its character, whether malignant or benign, was not possible to be determined in this manner. The above numerated symptoms, however, justified diagnosing the case as stricture of the bowels secondary to a new growth. A proper romanoscopic examination would have solved the difficulty and would have enabled a correct differentiation between malignancy and benignancy. Unfortunately owing to the social standing and nervous state of the patient and the anxiety of the attending physician to avoid as much as possible extraordinary manipulations or examinations, the employment of the sigmoidoscope had to be abandoned.

An abdominal operation upon this patient performed by Dr. William Francis Campbell disclosed in addition to a lower position of the stomach and the displacement of the transverse colon into the pelvis, a thick strong band of fibrous tissue, two inches broad, very much resembling a Jacksonian membrane. This was found at the junction of the sigmoid flexure and the descending colon holding the gut downwards and against the pelvic wall making it tense, immovable and causing a decided angulation and obstruction of its lumen. The rest of the abdominal contents were, upon exploration, found to be in a perfectly normal state. The patient made an uneventful recovery, with the disappearance of all her previous complaints.

In this case, while a surgical operation was indicated in either event, yet a careful proctoscopic examination of the lower bowel would have excluded malignant tumor as a factor and would have enhanced greatly the chances of an exact diagnosis.

Case III. (Referred by Dr. Ph. Oginz.) M. B., 24 years, male, Russian. His history is unimportant with the exception perhaps of a certain amount of gastrointestinal complaints and constipation. Habits—perfectly good. Patient consulted his physician some few months previous for frequency of his bowel movements, from five to six daily. These were usually of a semisolid consistency, at times

more liquid, never bloody or painful, but frequently mixed with mucus. He also experienced considerable loss of weight and gurgling sensations in the abdomen. A gastric analysis made by his physician disclosed a total absence of digestive acids. Hence the diarrheas were ascribed to the stomach condition and pronounced as gastrogenous. But the administration of hydrochloric acid even in large doses for the relief of the achylia gastrica did not seem to ameliorate his diarrheas. Upon examination, I found the patient exhibited a number of symptoms typical of individuals of the vagotonic type. His pupils were of different sizes, the left eye deeper situated in the socket than the right, his face flushed, his extremities cold and clammy, hyperhidrosis, dermatographism, pollakiuria, highly exaggerated reflexes, mucous colitis and eosinophilia.

In this case the diarrheas could have been properly accounted for by the peculiar constitutional state of the patient and his achylia gastrica, but a proctoscopic examination not neglected in this case revealed conditions which have been the true cause of his intestinal derangement, and which would not have been detected otherwise. A number of very fine ulcerated separated areas situated high in the sigmoid were thus detected. These were the cause of his diarrheas, as was shown by the disappearance of the patient's complaints upon the cure of his ulcers by proper dieting, the administration of hydrochloric acid in concentrated form and local applications.

Case IV. M. C., male, 40 years old, merchant, Russian. At the age of 16 the patient had typhoid fever; otherwise his history has no bearing upon his case. For the past six months the patient complained of constipation and vague gastrointestinal irregularities. These were ascribed by his attendant as due to gastric ulcer, and the proper dietary and mineral oil to overcome the obstipation were prescribed, but at a later period the patient began to suffer cramps in the abdomen and frequent and very urging evacuations of the bowels. These discharges were ill smelling, yellowish or dark colored and of an oily consistency. Careful examination upon consultation revealed nothing abnormal except a somewhat higher blood pressure (160 mm. Hg.), and increased skin and periosteal reflexes. His stomach contents after the usual test breakfast was

straw-colored, finely granular, 100 c.c. containing no blood or lactic acid, but pepsin, lacferment, and microscopical ingredients in normal amounts and appearances. The examination, macroscopic and microscopic, of the feces proved the presence of a large amount of pus and blood in addition to the normal varieties of food rests. The sigmoidoscope revealed the real etiology of his complaints. There were dry membranous hemorrhoidal knots in the lower part of the rectum and at about 10 cm. from the anus the whole mucous membrane encircling the gut at that position for about 3 inches wide was rough, ulcerated, injected, and studded with numerous papillary projections easily bleeding and quite sensitive to touch. The surface of this ulcerated area was covered with a purulent discharge. In this case, previously diagnosed as an *ulcus ventriculi*, the application of the proctoscope facilitated the correct diagnosis of the case and permitted the correction of an incorrect diagnosis.

I could cite from personal experience a number of similar instances demonstrating the value of thorough investigation of the lower bowel. These citations are not enumerated with the object of unkind criticism, but rather to emphasize the consequences of failure to examine the lower bowel properly. A brief description of the manner in which such an examination should be conducted seems therefore appropriate.

At first we will review a few points of the anatomy of the organs in question which are of practical interest. The rectum begins at the third sacral vertebra above and ends at the anus below. It is a tubular organ, concave in form and lies closely and parallel to the inner surface of the sacrum and coccyx. The rectum is practically devoid of freedom of motion, especially in its lower portion, and without peritoneal covering. Its length from sigmoid to anus is 11 to 13 cm. It may be divided into two distinct regions: (a) the *pars sphincterica*, 4 cm. long, the lower and smaller, which is surrounded by the external and internal sphincters and which plays the more frequent rôle in the pathology of the organ, and (b) the *pars ampullaris*, 7 to 9 c.m. long, the upper and larger portion, which is less frequently the seat of disturbances. When empty the *pars ampullaris* presents the appearance of a tube collapsed with its walls in opposition; when distended however it appears as a spindle-shaped affair measuring from 7 to 9 cm. in length, 4 to 6 cm. in

width and with a capacity of 250 cubic cm. In appearance the mucous membrane lining the rectum normally is perfectly smooth, pink in color, moist and with a lustre; there are no prominent blood vessels or mucus displayed upon its surface. In the upper, second or larger portion of the rectum two, sometimes three folds of mucous membrane project into the lumen of the gut. These are the valves of Waldeyer or Houston, the lower often being known as the *plica coccygea*, while the upper is called the *plica sacralis*. Somewhat smaller and less conspicuous is the fold marking the separation of the rectum from the sigmoid, the *plica terminalis*.

Adjoining the upper subdivision of the rectum is that part of the sigmoid which permits inspection by the sigmoidoscope. It may be well to make mention here that the entire length of the sigmoid cannot be examined in this manner; only its lowermost section, that extending from the *plica terminalis* upwards to the *plica labialis* of the sigmoid, is accessible to direct inspection, but not the segment beyond the last mentioned *plica labialis*. This circumstance arises from the fact that the gut, itself fixed, makes at that point a sharp turn or an acute bend, thus preventing the introduction of an instrument beyond this point. That portion of the sigmoid flexure accessible to inspection is about 20 to 22 cm. long; its mucous membrane appears to be thrown into numerous small folds, the *rugæ flexuræ*, wrongly termed by some authors valves. In all other respects the mucous membrane of this part of the sigmoid flexure is of the same appearance as that of the rectum.

With this short review of anatomical data we may now proceed with the description of the method of examination of the lower bowel. There are two of these: (1) the palpation or digital method and (2) the inspection or recto-romanoscopy. The digital method is of distinct value and should never be neglected, but the direct inspection method is the more important one. Numerous details which escape detection otherwise can be appreciated by the eye only. The color of the mucous membrane, its glossy appearance, its moisture, minute hemorrhagic spots, smallest erosions, the character of exudates, the presence of pseudo-membranes, the location of discharges, etc.—all these and other characteristics can be appreciated only by inspection and very rarely by palpation. Nevertheless, a digital examination helps in the determination of the condition of the mucosa, its thickness, its smoothness or roughness,

any swellings or new growths, etc. To detect the latter it oftentimes is necessary to change the position of the patient from dorsal to the lateral, while keeping the examining finger in the rectum, as the tumor may be situated within the posterior rectal wall, and when so hidden it is inaccessible to palpation. However, the changing of the position of the patient to the genu-pectoral oftentimes causes the posterior wall and inclosed tumor to gravitate downwards and to become accessible to palpation.

The other method of examination, that of direct inspection, is accomplished by special instruments. Valve speculæ are serviceable, but are of limited usefulness. The shortness of their blades does not permit inspection of parts above the top of the instrument; furthermore, the blades obstruct the view of the mucosa; the latter often bulges between the blades giving the appearance of congestion or the instrument stretches the bowel wall, making it appear paler than it really is. Another bad feature of valve speculæ is that the tissues become caught between the blades when the latter are being closed for the removal of the instrument, an occurrence which may cause considerable injury to the tissues and pain to the patient.

Of the numerous recto-sigmoidoscopes in use I find the one most serviceable and hence in personal use that of Schreiber as modified by Singer. This instrument seems to include all the good features of the others and is very solidly constructed mechanically. Another very serviceable instrument is the colonoscope of Heinrich Stern which is manufactured in the United States. Before rectoscopying the patient to avoid fecal matter from obstructing the view it is desirable to give the lower bowel a thorough cleansing. This is done best by the administration several hours before the examination of the common enema. It is important to administer this treatment several hours previous to the examination; otherwise the peristalsis induced by instrumental manipulations may cause the remaining liquid from the previously administered enema to descend from its higher position in the bowel during the examination, thus rendering observations unpleasant and even impossible. Nevertheless, there are occasions when it is desirable to study the natural condition of the mucous membrane with its exudates, mucus, pus, or other pathological products in the places of their several locations; the cleansing is then contraindicated.

As for other preparations none is required unless painful anal fissures, inflamed hemorrhoids or nervousness of the individual complicate the examination. Under these circumstances a 0.5 gram anesthesin or other palliative suppository is placed into the rectum before the examination. For the convenience of the examiner it is advisable to place the patient upon a high examining table. It is also necessary to put the patient in the genu-pectoral position, with his legs well separated and perpendicular to the table; his chest must rest flatly against the table and the spine bent well downward, the back muscles being thoroughly relaxed. This position has a great many advantages over the one formerly employed. Its value is based upon the identical principle underlying the same position when employed by gynecologists, namely the dilatation of the rectum by atmospheric pressure. Gravity is another important and advantageous factor of the genu-pectoral position. In this position the viscera of the abdomen and pelvis fall forward and downward toward the anterior wall and diaphragm, removing their pressure against the rectum, thus permitting distention of the latter by the inrushing air when the organ is opened by the introduced instrument. The view of the intestinal wall is then so complete that with good illumination the whole mucosa may be studied quite exactly. During the examination an assistant with his right arm encircling the abdomen of the patient should stand to the right of the latter in order to support this, while with his left hand the assistant should help the examiner by separating the buttocks when the instrument is being introduced. The tube of the proctoscope should be well anointed and by a slight screwing motion carefully introduced into the pars sphincterica for a distance of 3 to 4 cm. The panelectroscope is then attached and its light directed toward the lumen of the tube. The further insertion of the instrument is conducted under the guidance of the eyesight.

Because of the tone and grip of the external and internal sphincters in the pars sphincterica, the introduction of the proctoscope into the first portion of the rectum is attended by a certain amount of resistance, but as soon as this part of the bowel is passed and the next one, the ampullary, is entered the capaciousness of the latter is immediately appreciated. There the instrument can be swung around with perfect ease. While passing through this region we

may encounter possible obstructions from the valves of Waldeyer. To overcome this we must so swing the end of the tube as to glide it over the free extremity of the valves. At a distance of 10 to 13 cm. from the anus the termination of this upper portion of the rectum is reached as evidenced by the appearance of the plica terminalis. Passing that we land into the visible portion of the sigmoid. Here again, because of the narrower width of the sigmoid, the canal appears tight but further introduction is not resisted as markedly as in the anal portion of the rectum.

It is needless to state that all these manipulations must be executed most cautiously. The various curvatures and foldings of the bowel should be carefully followed; the patient is to suffer no pain except perhaps a slight discomfort; injury to the delicate mucous membranes should be avoided; and under no circumstances should force ever be employed. When obstruction is encountered gentle manipulation only must be utilized to overcome the difficulty. In this manner we are often able to examine the bowel to the furthestmost point of possible direct observation, the plica labialis.

When encountering bends of the walls of the gut, a pneumatic attachment, consisting of a rubber bulb and window, devised and used for many years by Tuttle, may be employed to straighten or separate these convolutions; but care must be taken, for reasons sufficiently plain, not to stretch the gut too much. As a general rule, this pneumatic attachment is not frequently required, but if employed it must be used with caution.

The observations of the bowel conditions are made during the introduction of the instrument as each portion of the mucous membrane presents itself to view, in the lumen of the tube. If covered by extraneous matter the membrane is carefully wiped clean with long cotton applicators before inspection. For further corroboration it is advisable to repeat the observations during the withdrawal of the proctoscope, especially of those places previously noted to be of interest. It is necessary to watch the gradations of the tube at all times so as to be informed as to what particular portion of the bowel is under immediate observation and especially whenever a point of interest is observed.

What is the normal appearance of the rectal mucosa and what are the indications for recto-romanoscopy? To answer the first

question: Normally, the mucous membrane of the rectum is perfectly smooth, velvety, somewhat shiny, of a rose color and not bluish as when congested or when complicated with internal hemorrhoids. The mucosa is also always moist, but not succulent, is free from mucus and reflects the light from the electroscope. In pathological states the appearance of the mucosa is decidedly different from that just enumerated. In answer to the second question, the indications for recto-romanoscopy, it may be said, that no hard and fast rules can be laid down as to the use of this method of examination. It is advisable in every case of tenesmus, rectal pain or discomfort, bloody evacuations, passage of mucus and pus, etc., to take resort to this exact method of diagnosis, unless such minor superficial affections as fissures, rhagades, piles, condylomata, etc., may account for the symptoms and may be detected by mere inspection of the parts. The enumerated symptoms usually indicate some serious disturbance located within the lower bowel.

Another use of the procto-sigmoidoscope is the localization of pathological processes. The determination of bleeding points in cases of hemorrhage, the localization of ulcers and abscesses, the identification of the seat of a stricture or stenosis, the detection of polyps and neoplasms—all these constitute positive indications for the use of the recto-sigmoidoscope. But not only is this method of value in the establishment of positive diagnosis, it is also frequently just as valuable for its negative evidences. By the recto-romanoscope we are enabled to exclude positively the presence of pathological conditions of the lower intestine when none are present in spite of complaints by the patient about this region, as is not uncommon among neurasthenics, hysterical and other individuals of a neurotic disposition.

COLON BACILLUS EXALTATIONS

(OBSERVATIONS AND IMPRESSIONS)

By B. G. R. WILLIAMS

Paris, Ill.

Some man has said that, "A colon bacillus is not a colon bacillus when busy in some other portion of the anatomy than the colon,"

an observation which I have come to believe correct, at least in part. The colon bacillus of water polluted with sewage and other colon bacilli found in nature as well as the resident of the large bowel usually come up to certain morphological, "physiological" and cultural standards; but the colon bacillus of the abscess invariably fails in one or more of them. Has it yielded these in part in the process of exaltation?

Before contrasting the several properties of the ordinary and exalted types, it seems pertinent to sound a warning. Do not be misled by the properties of a strain taken from pus and grown upon artificial media. Very likely it is now but an ordinary type and certainly is no longer pathogenic for the human. The best criterion is, of course, its activity in the tissues of the living human organism, and careful observation of its properties there.

Motility.—The colon bacillus found in pus often shows about the same degree of motility as the ordinary strains (we cannot say "nonpathogenic," for these inoculated may cause pus formation; I term them "ordinary" because they are not as yet sufficiently exalted to cause trouble without inoculation). In other words, motility is variable and may not be marked. It seems to me, however, that the bacilli of the kidney infections are often quite motile. At least the bacillurias of the Eberth and Escherich types are not likely to be differentiated upon the property of motility alone. The colon bacillus is often very actively motile in the freshly voided urine, whereas the typhoid bacillus may appear quite at rest. Many men are being misled by the old advice that the colon bacillus is nonmotile. This is more or less true with the ordinary forms, but the exalted colon bacillus is not a colon bacillus.

Form.—There is but little or no change of form in exaltation. That thread formation, however, is rarely or never seen in the colon rod of pus, is my observation. The exalted strain shows more "individuality." In standing urines there appears to be a distinct tendency to agglutination before precipitation. In the colon-typhoid group of bacilli increased motility and individuality appear to be concomitants of exaltation.

Odor Producing.—The colon bacillus has been termed the *B. pyogenes foetidus* because of the horrible fetors associated with

certain anal abscesses. This term is misleading, it seems to me. A foul smell is not present in the first of a series of these abscesses, and a bad odor of indol, amines and so on is likely to be of favorable prognostic import, appearing in the last of the series and suggests attenuation rather than exaltation. The idea is seducing that the bacillus has been overpowered and is reverting to a property of its ordinary strains. Moreover, the "permanently exalted" types of this group of bacilli, as the typhoid and paratyphoid members, are not associated with the liberation of offensive gases. In colon pyelitis, the urine is rarely foul. Yet at autopsy when the kidney is incised it is likely to loosen a very foul gas, showing that at death the bacillus quickly reverted to the ordinary form, and is, when exalted, a facultative saprophyte. In exaltation, therefore, the colon bacillus is likely to sacrifice its odor forming properties.

Acid Producing.—On proper media the colon bacillus is distinctly an acid producer. In the test tube azolitmin added in small quantities will be turned pink, than red. It has been suggested that in the living human being this bacillus gives rise to acid formation. The urines of colon pyelitis are invariably intensely acid. Of course, the acids may be provided by the diseased human cells, inasmuch as a high acidity is likewise noted in tuberculous kidney. In living connective tissues, however, we have no data which show that the colon bacillus elaborates acids. If so, these are rapidly neutralized or removed, for the pus is alkaline or neutral. While we are unable to positively state that this property is lost by exaltation, such appears to be the case.

For various manifest reasons it is impossible to determine whether or not other properties of the colon bacillus are lost by exaltation. Cultural tests in other animals prove nothing as susceptibility, hence degree and type of exaltation vary. Moreover, when removed from the living human or upon the death of the latter, the colon bacillus appears to revert easily to the ordinary type.

HOW IS EXALTATION EFFECTED?

We do not know how exaltation of the colon bacillus is effected. We have two theories concerning the situs of exaltation. The older is the ascending theory, urging that these infections are fecal inoc-

ulations, occurring when ordinary colon bacilli are passing or have passed the anus. But it seems to me that the hematogenous is the more plausible for the following reasons:

1st. Most bacteria at the anus are already attenuated or quite dead notwithstanding their enormous numbers. It is not likely that these would be easily exalted.

2d. Notwithstanding the frequency of statements to the contrary, I am certain that true colon infections are not especially frequent in the puerperium, and even if such were the case ascending infection would not be proved. In my observation they are more frequent in young women and even in babies (even where great care is taken with the diapers). They are frequent in the school teacher and shop girl forced to be on her feet for many hours, where they often pass as "cystitis," and in young married women especially where pregnancy is delayed. They are usually worse at the catamenial periods, and may disappear with the next pregnancy.

3d. We know that at least certain of these infections must be hematogenous. Ascending infection cannot explain the perirenal abscess or the pyelitis following skin abscesses.

4th. The colon infections do not appear to be strictly mucous membrane infections, as a rule. The colon bacillus appendicitis is more likely to be a perityphlitis than a catarrhal infection. The "pyelitis" is probably an involvement of the interstitial tissue of the renal medulla if my interpretation of sections is correct. Moreover, the perirectal and perirenal are the other usual ones, and these are unquestionably connective tissue infections.

FALLACY OF VACCINES

If what has been stated above is true, the fallacy of vaccine therapy will be noted at once. The exalted type changes immediately to the ordinary type after removal from the living human tissues or upon the death of the latter. Even as certain nonpathogenic properties are apparently regained so are other pathogenic (immunity producing) properties lost, and bacterins (vaccines?) produced from it vary in no way from those constantly present by virtue of the residence of the colon bacillus in the bowel, as well as his life's activities and death in that location.

THE RECOGNITION OF THE PRETUBERCULOUS STAGE AND THE EARLY SYMPTOMS OF TUBERCULOSIS

By ALBERT C. GEYSER

Professor of Physical Therapeutics, Fordham University Medical School;
Late Clinical Instructor in Radiography and Radiotherapy, Cornell
University; Late Lecturer in Electrotherapy and Radiography,
New York Polyclinic School and Hospital

New York

"Prevention is better than cure." If this is true, it seems that we ought to change our views as to the stereotyped text-book teachings of ready-made diagnoses. As students we were drilled to the observance of certain symptoms, and when these symptoms were present we were dealing with a certain disease. In other words, we were obliged to wait until the disease process was confirmed by its symptoms, then we made the diagnosis and started in to treat the disease.

That may be the practice of medicine, it may even be the cure of the disease, but it certainly is not prophylaxis. In order to prevent disease we must recognize signs and symptoms which herald the approach of the disease. Right here let me digress for a moment from the theme. Prophylaxis in its widest sense can never be realized until the present insurmountable wall that exists between the medical profession and the public has been broken down. How can we practice prophylaxis unless the public is educated and taken into our confidence? We must have the patients come to us before they are suffering from the stereotyped text-book symptoms. The tuberculous patient must not wait until the cough will not yield to ready-made cough mixtures bought over the drug counter. Neither must the physician wait until he can detect consolidations and cavities and find the tubercle bacilli in the sputum.

Doctor S. G. Ehrenreich, of the Montefiore Home for Consumptives, recently examined 3,310 cases for the purpose of discovering the time elapsed between the onset of the first symptoms as noticed by the patient and the seeking of medical advice. His investigation disclosed the following facts:

DURATION OF ILLNESS BEFORE MEDICAL ADVICE WAS SOUGHT

<i>Duration</i>	<i>Males</i>	<i>Females</i>
one month or less	100.... 5 per cent.	70.... 5 per cent.
one month to three	458....24 per cent.	308....23 per cent.
three months to six	590....30 per cent.	470....34 per cent.
six months to one year	430....22 per cent.	286....22 per cent.
one year to two years	244....13 per cent.	161....12 per cent.
two years or more	108.... 6 per cent.	85.... 4 per cent.

Fifty per cent. were sick at least six months before seeking medical advice. Fifteen per cent. of all of these cases were advised by their physician that the lungs were not affected. Doctor Ehrenreich then states, "I can safely say without any contradiction that the physicians' neglect in not properly examining and diagnosing pulmonary tuberculosis is responsible for more than twenty per cent. of all of our advanced cases."

I am quite sure that if the family physician explains to his patients that it is a far greater achievement to prevent than even to cure a disease, there will be a just and proper appreciation on the part of his patients.

For the sake of convenience I shall arbitrarily divide tuberculosis pulmonalis into the pretuberculous and the early stages.

If we would practice prophylaxis in tuberculosis it must be done in the pretuberculous stage. Before this is possible it is essential that a certain few facts concerning this disease must be appreciated.

There are certain tissues in the body that are invaded by the tubercle bacilli in preference to all others. They are the apices of the lungs, the ends of the long bones, articulations in general, the glands and the skin. While all of these tissues differ anatomically and physiologically, they have one thing in common, that is physiological anemia.

To have a suitable soil for tuberculosis we must have anemia. Having anemia we must have the bacillus tuberculosis taking up its habitat there. Again, it is not the bacillus tuberculosis that kills the patient, but a certain toxic material given off by the bacillus. This toxic material causes further anemia whenever it comes into contact with tissue cells, hence further spread of the disease. We

have three points to bear in mind, the anemia, the bacillus and the toxic element. Since the existence of anemia prepares the culture medium and the toxines increase the same and eventually overcome the individual, and as the bacillus is only the necessary intermediary agent, it behooves us to recognize and prevent anemia, to recognize and to overcome the manifestations of the toxic elements upon the system and at the same time to pay as little attention as possible to the bacillus tuberculosis.

Starting out upon these admitted premises let us look for the earliest symptoms of the pretuberculous stage.

These symptoms are subjective and objective. The subjective symptoms cause the patient to consult the doctor for nearly every thing else except tuberculosis.

1st. General malaise and fatigue. The patient tires easily, his appetite is capricious, he especially avoids fat and cream, nothing seems to taste just right, he becomes irritable and moody, he thinks that he needs a tonic or a stomachic, his hours of labor seem too long, his work is becoming distasteful to him, he longs for a change, he cannot stay at home because every one thinks him peevish. This is the early irritable effect of anemia and toxemia.

2d. Upon awakening in the morning there seems to be an unusual amount of mucus present, he is obliged to clear his throat, he hawks and expectorates, but he remembers having been in a draft of fresh air and so "caught cold." He either partakes of the family cough remedy or the druggist "fixes up something." Cough ever so slight must be accounted for.

3d. Loss of weight. For some reason the patient does not seem to fill out, he remains lean, long and lanky; for his age he is taller than his playmates, but his weight remains about ten or more pounds below the normal.

4th. The complexion is "perfect," the skin is white, does not tan, but burns in the summer. If it happens to be a girl the complexion does not require "making up," especially late in the afternoon; the eyelashes are long and abundant, the pupil frequently dilated and the eye bright, the hair growth almost luxuriant, but each hair is thin and dry, giving it that much desired fluffy appearance. The fingernails are long and shapely with a bluish white background.

5th. Small pulmonary hemorrhages or sanguinous expectorations

especially occurring in women with a tendency to lessening of the menstrual flow must be construed as significant.

6th. Rapid pulse. As soon as the system begins to absorb the toxic products of the tubercle bacillus there ensues a reaction on the part of the system to overcome this toxemia. The result is a quickened pulse rate. A daily pulse rate of eighty-five or more without other discoverable causes becomes suspicious.

7th. Increased temperature. Hand in hand with the increased circulation we have the increased temperature. It is far better to be guided by the daily variations between the minimum and the maximum than by the temperature per se. The normal temperature differs with each individual, but if the daily variation exceeds one and six-tenths degrees F. it should arouse our suspicion. The daily variation in a tuberculous subject amounts to from two to three and a half degrees F.

8th. Progressive loss of weight. A patient with incipient tuberculosis giving the two previous symptoms of necessity is subject to hyperoxydation, hence the systematic loss of weight. This loss of weight is entirely out of proportion to the food intake or manner of labor performed. Such patients abhor fats, they not only fail to assimilate it, but they actually eliminate it.

Fat is free in the circulation, the absorbents are taking the fat from the various deposits into the circulation for the purpose of aiding oxydation or the production of body heat. Free fat can be demonstrated in the blood in most cases.

9th. Litten's phenomenon. With even the slightest tuberculous infection of the lungs, the diaphragm upon the affected side does not make its full excursion during inspiration or expiration. In a good light, a shade or wave-like motion can plainly be seen to lag behind its fellow of the opposite side. From a physiological viewpoint the reason for this is obvious.

10th. Supraclavicular retraction. As soon as tubercles have formed or are forming in the upper part of the lung, inflammatory adhesions develop. As a result of this the supraclavicular fossa on the affected side is markedly influenced upon deep inspiration and expiration. The difference between the two fossæ is very noticeable.

These are some of the early clinical signs of pulmonary tubercu-

losis. Whenever a majority of these are present, the case should be considered as in the pretuberculous stage, if the patient shows the minority of these symptoms then laboratory aid must be sought.

1st. The tuberculin reaction. Whether this responds to the cutaneous, the subcutaneous or conjunctival tests is immaterial. Reaction means that there is or recently has been a tuberculous process somewhere in that patient. As to the choice of these methods too much value must not be placed on either of them; in Müller's medical clinic at Munich the ophthalmic test is entirely forbidden as not only useless, but also dangerous.

The von Pirquet reaction. V. I. Glitschikoff studied 148 cases and showed that in tuberculosis the intensity of the reaction is in inverse relation to the severity of the disease. As the disease progresses the reaction becomes weaker, often disappearing in the last stages. In other words, the system is no longer able to respond or react when the cells have been completely placed under the effect of toxins from the tubercle bacilli.

The subcutaneous method is contraindicated in the presence of fever, hemorrhage, definite physical signs or the bacilli in the sputum. By either method a positive reaction can have but very little diagnostic value as far as early clinical tuberculosis is concerned. At its best it can only be confirmatory with other pre-existing symptoms.

If the process is latent and the local lesion cannot be determined such a patient should be considered as tuberculous, but he should not undertake any special therapeutics. Hygiene and prophylaxis are his safeguard. Nature seems to be overcoming his disease and we have not yet been able to improve upon the "*vis medicatrix naturæ*."

2d. The presence of the bacilli in the sputum. Tubercle bacilli never appear in the sputum unless softening of the focus has taken place. It is, of course, *prima facie* evidence of infection. If the greater majority of the early clinical symptoms are absent, but the laboratory findings present upon repeated examinations, such a patient should be considered as being in the first stages of the disease. When a majority of the clinical symptoms are present plus the laboratory findings, the family history good, the physical condition at par and the hygienic environment suitable, such a

patient should be considered as in the early second stage. When all of the pretuberculous clinical manifestations are present plus laboratory findings the patient is in the late second stage.

It matters little as to time, whether these conditions have been present for one month or several years. Some patients never get to the first stage, others live for years in the second stage, while still others succumb to the third stage a few weeks after the onset.

The recoveries under modern methods (physical and diathermic treatment) are ninety per cent. and over in the pretuberculous stage, eighty-five per cent. in the first stage, and, as Doctor Van Rensselaer of the Albany tuberculosis camp has shown in his presidential address last May, sixty-nine per cent. in all but the very last stages.

It may be true that some of the cases diagnosed as in the pretuberculous stage would never develop the real disease. When we reflect upon the fact that every child before the age of twelve years has been infected, that ninety-five per cent. of all cases coming to autopsy show signs of healed tuberculous lesions, that one out of every eight persons dies of tuberculosis, it is safer to run the risk of treating an occasional or even quite a number of such cases and restoring them to health clinically, than to run the risk of letting a single one escape and develop the disease beyond human aid.

In tuberculosis as in cancer when the diagnosis is so positive that it can be made by the laity, it is apt to be too late for efficient therapeutics.

THE EARLY DIAGNOSIS OF INCIPIENT PULMONARY TUBERCULOSIS.

By CECIL C. LAWHORN

Physician in Charge, North Side Children's Tuberculosis Clinic
Milwaukee, Wis.

Tuberculosis in some form has been found to be present in 90 per cent. of children up to 15 years of age. Calmette says that 90 per cent. of children from 5 to 15 years of age, and from 91 to 97 per cent. of young people above that age are affected with some form of tuberculosis. Von Pirquet states that 90 per cent. are infected in the first year of life.

In Fishberg's series of 692 children under 15 years of age, examined medically and by the von Pirquet test, belonging to 317 families where one or both parents were tuberculous and had applied for relief to the United Hebrew Charities of New York in March, April and May, 1913, 67.23 per cent., or 465, were found to be tuberculous.

Comby reported 638 cases in 1675 necropsies upon children 15 years old or less from the combined hospital statistics in Paris. Between the 10th and the 15th year of the series, 71.23 per cent. were tuberculous.

Leroux, of Paris, says that from his radiograms, it is evinced that the base or middle part of the lung is in the majority of cases the primary seat of inoculation in infants and young children, but that in children over 10 years of age and in adults it is the apex which is usually involved.

By radiography carried on for two years it is possible to show the evolution of glandulo-pulmonary tuberculosis in three stages: a. Pulmonary infection of some part of the lung and unilateral adenopathy. b. Tracheo-bronchial adenopathy more or less latent. c. Reinfection of the apex and evolution of chronic pulmonary disease.

A diagnosis of incipient active pulmonary tuberculosis involvement is greatly to be desired and from the recent light thrown upon childhood infection, the examining physician should suspect phthisis in every child or adult presented for examination who may have any suspicion of lung disease or have symptoms of anemia, malnutrition, poor sleep, gastric disturbances, diarrhea, tachycardia, pains in chest or abdomen, lassitude, loss of weight, and in children a failure to gain weight, hoarseness, fever and tracheo-bronchial adenopathy as evidenced by d'Espine's sign, positive tuberculin skin reactions and radiography.

A. F. Beifeld in a recent article in the ARCHIVES OF DIAGNOSIS for October, 1914, "An aid in the early diagnosis of pulmonary consumption," mentions the apical percussion methods of Goldscheider and Krönig, the muscular spasm phenomenon of Pottenger, acromial lagging, pupillary inequalities, myoidema and acromion auscultation recommended by Abrahams and Magida. The latter method in the diagnosis of incipient tuberculosis which has had such wide publicity of late, I have found of no value owing to the inability of

the examiner to determine the pathological from the physiological signs present in probably normal individuals.

To bring out the fine diagnostic moist râles on auscultation, Beifeld mentions further the well known deep inspiration, sharp coughing, a deep inspiration after the patient has counted as long as possible in a single breath, administration of potassium iodide, etc. In addition he describes a new method, "The whispered voice method." The patient is instructed to whisper in an emphatic manner in a single breath, "One, two, three," several times following which he is to inspire deeply. (The latter he does spontaneously.)

I am able to verify this method as being quite effective, having used the same since 1910, when I observed the phenomenon by accident in testing whispered pectoriloquy in the Tuberculosis Clinic of the Presbyterian Hospital in New York. In addition to and in conjunction with this I go further. I instruct the patient to count repeatedly in whispers "One, two, three," six or more times, in the same outgoing breath and then to cough also in the same breath, taking care not to inspire at all before the cough, then to inspire deeply, immediately after the cough. The patient will always spontaneously take a deep inspiration after the cough.

If the fine râles sought for are constantly present and not dissipated on coughing followed by deep inspirations or are elicited regularly after the continued whispering, coughing and inspiration combined, the conclusion of a positive diagnosis is justified.

The diagnostic factor is that if the fine moist râles are transitory or disappear by this or any of the auscultatory methods, the condition is not of a tuberculous nature as is exemplified in a bronchitis, where upon the first examination more or less suspicious râles are discovered, but upon repeated deep inspirations or coughing they disappear temporarily or permanently, while in pulmonary tuberculosis the opposite is true.

An experience of several years has strengthened my belief in the correctness of my observations. I have found the methods valuable and effective in the examination of children who will not breathe satisfactorily, in adults who cannot or will not breathe in the way desired, and in very muscular men whose normal muscle sounds interfere with the respiratory sounds. I remember the case of a young German, 21 years old, a seaman. He was very robust, had

large, powerful muscles, and was a picture of health, but had a suspicious cough. When instructed to breathe deeply, the muscle-sounds completely overshadowed the respiratory sounds; on account of the great muscular development percussion was also unsatisfactory. The whisper and cough method was used, which permitted the râles to be heard, a positive diagnosis was made and a month or two later a positive sputum was obtained.

The presence of the fine râles, whether at apices or elsewhere, in patients suspected to have pulmonary tuberculosis (when non-tuberculous conditions which may account for them can be excluded) afford the earliest positive signs of pulmonary tuberculosis.

In an endeavor to explain the phenomenon brought out by the whispering method, I think that the strain of continued whispering in a single outgoing breath causes by reflex action an overproduction of moisture, especially in the finer bronchioles and alveoli; then by complete collapse of the walls of the alveoli due to the cough and immediately followed by deep inspirations, the consequent separation of the moist, agglutinated, diseased walls of the air sacks, and the air rushing through the moist bronchioles, these two factors produce the râles, whereas in a simple bronchitis the excessive moisture and pathology is only in the bronchioles and bronchi, forced breathing clears these air passages and the râles disappear, but in incipient phthisis the râles are brought out more distinctly and remain permanently.

TETANISM

By HERMAN B. SHEFFIELD

New York

The two-months-old baby was entirely normal at birth. He weighed eight pounds and thrived nicely the first two weeks of his life while nursed by his mother. Owing to financial distress, however, the mother was soon compelled to work out by the day and to entrust the care of her baby to her old mother, who fed it on a milk mixture of her own design. The baby soon began to fail, and accordingly the feeding underwent almost daily changes to suit the good judgment of the numerous neighbors who owned sturdy babies. Finding, finally, that all the well-intentioned sug-

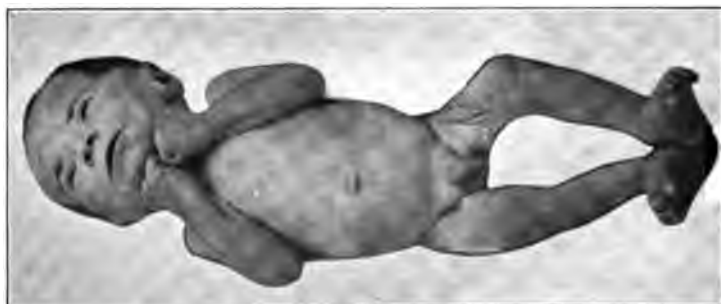


FIG. 1.
Tetanism during acme of spasm.



FIG. II.
Tetanism during partial relaxation
of spasm.

TETANISM
Herman B. Sheffield

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gestions proved of no avail, the mother concluded to try a doctor. When the baby came under my observation at the hospital he weighed four and a half pounds. He was the very image of a marasmic baby. He had a voracious appetite, but almost invariably vomited after feeding. The stools were frequent, green and filled with undigested particles of food. He suffered from colic especially soon after feeding, was restless, cried and whined pitifully, and slept poorly. The anterior fontanelle, the eyes and cheeks were sunken, the nose and chin were pointed, the abdomen was retracted, the skin wrinkled, in some places hanging in folds, and adding to this the earthy pallor and senile expression of his face, the poor creature was a sight dreadful to behold. As a further addition to his misery he was suffering from a symptom-complex which a few years ago* I ventured to describe as *tetanism*. This is a peculiar form of continued muscular hypertonicity occasionally observed in very young infants with markedly lowered vitality, be it as a result of prematurity, syphilis or chronic gastroenteritis. The onset of the spasticity is fairly rapid, and in severe cases, when fully established, the posture assumed by the patient is pathognomonic (see Fig. 1). The head is moderately retracted, the facial muscles are contracted, the jaws are firmly set together, the forearms are flexed upon the arms and the hands are tightly clinched, so as to form firmly closed fists. As a rule, the legs are bent angularly and the feet either overlap each other or are arched. The muscular contractures relax off and on (see Fig. II), more especially during profound sleep, but never subside entirely. The hypertonicity increases on handling the baby, but it never interferes with feeding. With improvement of the general health of the baby, the contractures gradually disappear.

As can be noted from the accompanying illustrations tetanism is a typical clinical picture easily to be differentiated from similar spasmodic affections. On the first examination of the patient we may suspect either tetany, tetanus or eclampsia, but on careful analysis of the symptomatology of these affections, the erroneous impression can readily be dispelled. Tetanism differs from tetany by its more gradual development and almost continuous persistence for several months; any kind of handling of the baby increases its

*Arch. of Pediatrics, Aug., 1910.

muscular hypertonicity, while in tetany the attacks may be brought about or aggravated only by pressure upon large trunks of nerves or arteries (Trousseau's phenomenon), electric excitability (Erb's phenomenon), or irritation of the facial nerve (Chvostek's sign). Tetanus is an acute disease, preceded by an infection, as a rule accompanied by difficult deglutition and respiratory embarrassment and usually ending fatally within a week. Eclampsia infantum occurs in attacks and is associated with loss of consciousness. In the same manner we can promptly exclude so-called meningismus; moreover, none of these spasmodic affections of infancy ever give rise to the characteristic contractures of the extremities just described and illustrated.

I. CAVERNOUS ANGIOMA OF THE LIVER IN A BABY SIX WEEKS OLD

II. CONGENITAL ABSENCE OF ALL ABDOMINAL MUSCLES

By ARTHUR STERN

Attending Physician to the Children's Wards, St. Elizabeth Hospital and the
Elizabeth General Hospital

Elizabeth, N. J.

Case I. Muriel M. Six weeks old, was sent into my service at the Elizabeth General Hospital through the kindness of Dr. Horace Livengood.

The mother, who is twenty-five years old, had three children of whom the first died, the second lives and is healthy, and this was born after a pregnancy during which the mother was anemic and suffered from varicose veins.

The child was nursed for two weeks and then put on artificial food. On Oct. 22d, the mother brought the child to Dr. Livengood's office saying that for the past few weeks the stomach of the baby had been growing larger and the rest of the baby thinner. There was very little digestive disturbance, no vomiting and occasionally a green, lumpy stool. The child, after examination by Dr. Livengood, who found a large tumor in the abdomen, was then sent into my service at the hospital.

THE ARCHIVES OF DIAGNOSIS



CASE I

CAVERNOUS ANGIOMA OF THE LIVER IN A BABY SIX WEEKS OLD
Arthur Stern

THE ARCHIVES OF DIAGNOSIS



CASE I
CAVERNOUS ANGIOMA OF THE LIVER IN A BABY SIX WEEKS OLD



CASE II
CONGENITAL ABSENCE OF ALL ABDOMINAL MUSCLES
Arthur Stern

The examination showed a greatly distended abdomen with a large, hard tumor coming from under both ribs and extending deep into the pelvis, and in the right lower side an indentation could be made out. The X-ray picture, after a small bismuth meal, showed the stomach to be normal and a dense mass in the abdomen.

The child stayed at the hospital only a few days and was then taken home, where it died Oct. 31st.

Dr. Livengood was able to perform a partial abdominal autopsy and found the tumor to be the liver. A part of it was removed and examined by Dr. J. H. P. Conover, pathologist to the hospital, who pronounced the tumor to be a cavernous angioma. There was very little liver tissue present, but large cavernous cavities lined with endothelial cells.

Liver tumors of this nature are exceedingly rare, and I have been able to discover only five cases reported in the literature.

Hippel, Philipp, Yamagiwa, and Nakamura have described tumors of mesoentodermal and mesoectodermal character of the liver.

In these five observed cases, entodermal adenomatous and even carcinomatous (Yamagiwa) formations were found intersected with myxomatous, fibrous, cartilaginous and even osseous formations.

The description of these rare cases is found in the Handbook of the Pathological Anatomy of the Infantile Age of Bruening and Schwalbe.

Case II. Baby N., male, was born as the third child of a family, where mother and father are healthy and also the first two children.

I saw the child immediately after birth. The abdomen formed a large bag, and with each respiration some of the abdominal contents were aspirated into the thorax. As far as I could feel, there was no diaphragm present and the abdominal wall was almost transparent, but contained large pulsating blood vessels. These vessels ran mostly into the umbilicus, but communicated with each other and pulsated. Immediately after birth the child passed free blood instead of urine and kept on bleeding until it died, forty-eight hours after birth. An autopsy was not permitted.

Apparently this case belongs into the large group of "Monstra per defectum." The interesting feature is the communication of

the blood vessels with the uropoetic system and the metaplasia of the abdominal wall with a highly vascular tissue.

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“PSYCHOANALYSIS” A WORD CAPABLE OF WIDE
USEFULNESS

By J. MADISON TAYLOR

Professor of Non-Pharmaceutic Therapeutics, Medical Department,
Temple University
Philadelphia

Psychoanalysis is a term full of meaning, signifying the process of unravelling subversions of consciousness and serving as the first step toward setting them in order.

Language suffers strange parodies while acting as vehicle of thoughts and purposes for sects dominated by vagaries of belief or feeling. The word “Psychoanalysis” has become thus burdened with hidden, unsavory wholly unfair mismeanings. It probably cannot be expurgated, but its essential meaning can, and should be restored and properly applied.

The Freudians have set the world astir by re-enacting a sort of Phallic revival; a reversal to earlier concepts of hysteria. “Psychoanalysis” is now nearly as familiar as “Urinalysis,” it could be made to become nearly as useful.

All who assume responsibility in ministering to psychopathies make, and long have made, use of psychological analysis in searching into the intricacies of mental perturbations. Psychogenesis of clouding of the consciousness was well known in ancient days, and good means were then employed to disencumber and free the sufferer from its effects. The clouds come from diverse sources and

avenues, chiefly sickness of the feeling tones; disharmonies between creature and environment. A life history has ceased to flow along normal channels; the subject has become quagmired, has wandered into strange ways; in short got lost; less from not knowing how to find the way than by misinterpretation of landmarks by reason of morbid pre-occupation and a growing terror of harmless objects; to frantic anxieties to get somewhere, forgetting that it would be easy enough if only primary laws of progression were observed.

A life history is thus turned aside from the even tenor of its way; then a guide is needed to follow after, to pick up the trail, interpret the divagations, hunt up the wanderer, reassure him and put his feet upon the right road and keep them there till power is regained to start afresh and work out life problems with confidence and awareness of one's powers.

The Freudian guide would have us believe that all such wanderings in the maze are due to sexual shock received during earliest glimmerings of adolescent development. He would insist that "libido" is a force which dominates each and every phase of human purposes, existence, faith and act.

Where, we may well ask, do the organic, toxic and other acquired causes and their conditions come in; not to mention the underlying developmental hypoplastic factors so common?

It is customary for any one ministering to a disordered mind to make systematic search into all the mental chambers, open and shut, patent or secret, leaving nothing undisclosed.

That process may be called a *Psychoanamnesis* (a contribution to picturesque verbiage). Thereupon we proceed to group the correlated findings together with due regard to perspective and values by *Psycho-synthesis* (another offering to descriptiveness), and are in a position to enter upon the task of *Psychoanalysis*.

Next in order is to form expert judgments, opinions on the significances of data obtained, and to reach conclusions on their nature and bearings, reaching a *Psycho-diagnosis* or the better term contributed by Boris Sidis, *Psychognosis*.

Now we are in a position to formulate procedures for rescuing the distressed or confused person from effects of personal errors, of act, of ideation, of emotivation, of hurtful self-estimates of his or her own mischievous or disabling fancies or faulty evaluations

of somatic possessions; thereupon proceeding to rehabilitate the dissociated elements of consciousness and apply the now popular and widely discussed measure known as *Psychotherapy*. This may seem a circuitous route to follow; it is well fortified by high sounding words.

Few or no advances in clinical medicine are so useful as the changing of a disorderly mind into an orderly efficient mind. It is not a modern discovery that the mind (or psyche which conveys a larger significance) is frequently affected so seriously as to resemble "madness" or insanity. The earlier observers recognized and appreciated the condition; some of them made illuminating observations and recommendations, helpful to us moderns, provided we interpret them aright. Moreover they supply clear vision as to the prevalence then, precisely as now, of mixed instances of somatic and psychic disorder.

Most, if not all, human ailments are complicated by personal misinterpretations of somatic derangements and diseases.

In determining means for complete and permanent relief or cure, the wise practitioner never omits to keep the factor of mind control in the foreground of his purpose.

This is true not only of protracted disabilities, but likewise of acute states. External stimulations, especially when excessive, amounting to cell irritation, or sensory disturbances, involve also anxieties about unknown possibilities. Fear effects are thus often transferred from, or to, those in the domestic or industrial circle. These anxieties may disturb mental equipoise to a degree not immediately apparent, nor determinable by commonly known means, yet elements of disaster are there, growing insidiously, or passing quickly, as may be decreed.

A group of morbid mental conditions is constantly presenting to every experienced practitioner where the word in season, spoken with wisdom, tact and force, limits disabilities effectively no matter what vagaries of medication or "rational procedures" have been, or shall also be, employed.

Many an obscure medical man, moreover, is the best of counselors, the volume of whose practice is by no means an index of the efficacy of his treatment. So unobtrusively yet radically does he make well there is no room for graphic demonstrations by pandering

to human weaknesses and vanities which enable a bolder one to appear a wonder worker, as shown by crowded offices and bewilderingly impressive methods. These last by their mass and glitter cause poor fools to stand amazed at his "business" and pray to be numbered among his wealthy and numerous clients.

These honest, capable clinicians often possess unappreciated gifts; sympathetic, broad, analytical minds, fortified by sincere desires to help those who are heavy laden, to lift up the weakhearted, to set the feet of the confused and discouraged on the right road to efficiency.

In brief, they employ psychoanamnesis, psychoanalysis, psychosynthesis, psychodiagnosis, and psychotherapy, unwittingly, yet effectively.

The followers of Freud contribute to the elucidation of vexing problems while engaged in a form of special pleading which closely resembles verbigeration in a contracted mental field. They give the impression of being sustained by strange waves of feeling, wishes and belief, rather than by a strictly scientific endeavor.

The enthusiastic admirers and devoted followers of Freud (with modesty all their own) claim that there is no other single factor in mental life so dominating as sex; no other that has associated with it such tremendous emotional values, which so predominate over even the instincts of self-preservation; that it is to quote Ernest Jones, "the great fundamental, highly emotionally valued instinct, which spreads out and touches every part in the psychic life, and which is manifested quite as universally in the activities and institutions of the social system."

We are besought by these gentlemen to accept their opinions unreservedly, exhibiting faith that they have vouchsafed to them special advices from some unerring source of power.

They show, however, an undue eagerness to demonstrate the universality of their postulate that "libido" is paramount which savors of a suspicion that it is not wholly provable.

One good thing they have done, given us a good word in psychoanalysis provided we put this word where it belongs, and not where they would keep it.

Progress of Diagnosis and Prognosis

GENERAL METHODS OF EXAMINATION—SYSTEMIC AFFECTIONS—DISORDERS OF GENERAL METABOLISM

The Renal Function in Nurslings and Older Children—A. BOSLER, *Zeitschr. f. Kinderheilkunde*, Vol. XI, 1914, Nos. 5 and 6.

Older children with renal disease had no markedly different renal activity than children with healthy kidneys. In some of the nurslings with healthy kidneys the sodium chlorid retention was remarkably high. This was probably the result of sodium chlorid hunger. MILL.

The Urinary Secretion in the Nursling—ENGEL, *Deutsche med. Wochenschr.*, Nov. 12, 1914.

A properly nourished nursling imbibing about 800 cc. liquid per day micturates on the average 25 times during the 24 hours. When ingesting from 1200 to 1600 cc. of liquid daily, an occurrence which is quite frequent, the number of micturitions may be increased to 60 or even 70 during the 24 hours. Generally speaking, the evacuation of the bladder ceases during sleep. During the day and when the muscular and nervous systems are most active, the urinations are most frequent. Most micturitions discharge from 10 to 20 cc.; the largest amounts are generally voided during the night, from 50 to 60 cc., and exceptionally from 70 to 90 cc. MILL.

Diastatic Property of Children's Urine—W. B. McCLURE and P. S. CHANCELLOR, *Zeitschr. f. Kinderheilkunde*, Vol. XI, Nos. 5 and 6, 1914.

The diastatic quality of the urine of children increases with the age of the children. It is possible that rachitis and chorea contribute toward augmentation of the diastatic property. MILL.

Acute Nephritis in the Nutritive Disorders of Nurslings—F. FRANK, *Archiv f. Kinderheilkunde*, Vol. LXIII, Nos. 3 and 4.

Conclusions derived from the observation and study of 22 pertaining cases. Acute nephritis does not occur frequently in the nursing period of infants. Acute nephritis in the nursling is mostly exudative in character and presents frequently a hemorrhagic tendency. This must be ascribed to the abnormally great permeability of the blood vessels during the first year of life. Etiologically, all types of infection and especially nutritive disturbances play a marked rôle. MILL.

Parenteral Metabolism—A. DEMBICKI and J. LÖWY, *Deutsches Archiv f. klin. Medizin*, Vol. CXVI, Nos. 5 and 6.

The parenteral metabolism is in part dependent upon the numerous fermentative processes, which are the expression of the function of the most varying organ-cells. The leukocytes are among the most important of these cell forms. Apart from phagocytosis, antitoxic, oxidative, reducing, and fat and albumin splitting properties must be ascribed to the leukocytes. After the period of digestion an increase in leukocytes can be demonstrated. Such increase is, however, denied by some authors. Authors noted in 134 cases an increase of leukocytes after digestion in 53 cases, a decrease in 49, and no change in 32 cases. Leukocytosis existed in 23 cases of diminished, and 16 cases of unchanged serum-concentration.

WESTERN.

Ehrlich's Aldehyde Test for Urobilinogen—O. H. ROBERTSON, *Cal. State Jour. Med.*, Feb., 1915.

A positive test is of very little value on account of the fact that it appears in such a relatively large number of conditions having no apparent relation to liver function. One negative test does not rule out the possibility of a positive test appearing later on. A persistently negative test is of more value than a positive, but in view of the lack of clinical data, it cannot be said to exclude a pathological liver condition.

SACHS.

Creatin and Creatinin Excretion in Diabetes and Nephritis—D. LAMPERT, *Zeitschr. f. klin. Medizin*, Vol. LXXX, Nos. 5 and 6.

Author made his investigations with the aid of the Authenrieth-Müller method. Healthy persons who ingested neither meat nor bouillon excreted between 0.9 and 2.4 grams creatinin per day. Most individuals excreted between 1.2 and 1.5 gram creatinin. Creatin could be but rarely demonstrated; when it occurred it was present in traces only. In diabetes, creatinin was either eliminated in less than normal amounts, or in amounts which approached the lowest normal limit. The amount of creatinin in all the diabetic cases generally was below 1 gram. In some of the graver cases, 1 gram or a trifle more was found in a few instances. In 5 cases of nephritis a diminution of the excreted creatinin was ascertained. This was the case when diuresis was not diminished and the renal function was but slightly lowered. Creatin was present in minute amounts in one case of marked renal insufficiency. In two cases of diabetes with synchronous renal sclerosis creatinin was excreted in particularly small amounts. There is a parallelism between the creatin excretion and acidosis in instances of grave diabetes. Thus, it is possible that the creatin excretion is also the result of the intermediary metabolism, i. e. that creatin is the lower, creatinin the

higher product of metabolism. The creatinin excretion may be employed in functional renal diagnosis.

WESTERN.

Abderhalden's Serum Reaction of Liver Tissue in Alcoholists—E. MARTINI, *Deutsche med. Wochenschr.*, Dec. 10, 1914.

In 9 alcoholists, the serum reacted distinctly positive with liver tissue 7 times. Of these, 4 had demonstrable hepatic enlargement. It is possible that the serum reaction may facilitate the early recognition of alcoholic liver affections.

MILL.

Psychotic Uremia and its Mixed Forms—E. REISS, *Zeitschr. f. klin. Medizin*, Vol. LXXX, Nos. 5 and 6.

The psychotic type of uremia is characterized by conditions of marked confusion, by illusions and hallucinations, and deep coma. These conditions are often of a very transitory nature, disappear and reappear, frequently a few times during the day. The affection need not be accompanied by a pronounced disturbance of the external renal secretion. In two of author's cases typical sclerosis of the cerebral arteries was found at the necropsy. The mixed types of this form of uremia may present manifestations that occur in any of the groups of uremia; often their manifestations are not characteristic. In the asthenic type of uremia there is a more or less complete obstruction of the renal passage, resulting in augmentation of the retention nitrogen. In convulsive as well as in psychotic uremia such renal disturbance cannot be demonstrated. The substances giving rise to the latter types of uremia are not retained in the body by reason of the diminished excretory ability of the kidneys. Concerning the place of their production and their nature we possess as yet no information. The psychotic form of uremia is more or less connected with the sclerosis of the cerebral arteries that has been demonstrated in many of the pertaining cases. Still, the clinical picture of psychotic uremia is by no means identical with that of sclerosis of the cerebral arteries and should be differentiated from it.

WESTERN.

Lymphocyte Increase and Altitude—M. E. STAINS and T. L. JAMES, *Arch. Int. Med.*, Sept., 1914.

At an elevation of 6000 feet there is an increase of the larger lymphocytes of at least 20 or 30 per cent. in both man and monkey. The total white blood cells per cubic millimeter are approximately the same at sea level and at an altitude of 6,000 feet, namely about 7500. The red corpuscles increase by 22 per cent. at an altitude of 6000 feet.

SACHS.

Preliminary Stages of Diabetes—P. BERGELL, *Deutsche med. Wochenschr.*, Dec. 17, 1914.

Author advances the following conclusions: The dissolving property of the human urine for cupric hydrate— $\text{Cu}(\text{OH})_2$ —is not

caused by a small content of glucose. The reduction property of normal urine is likewise not due to glucose nor to uric acid per se. The urine of persons with normal carbohydrate metabolism, diluted to a specific gravity of 1012, exhibits a slight copper dissolving quality when small amounts of carbohydrates are ingested, and the specific gravity of the urine does not exceed 1020. The copper dissolving property of the urine is occasionally much augmented in children. In relatives of diabetics it is increased in two-thirds of the cases. A certain percentage of these cases, especially those with the most pronounced reactions, should be looked upon as being in the preliminary stages of diabetes. The copper dissolving property of the urine seems to be dependent upon aldoses and ketoses that possess a lower molecular weight than glucose. The increased copper dissolving property disappears when the carbohydrates are withdrawn. When more carbohydrates and glucose are ingested, the reaction becomes stronger, and, in the preliminary stages of diabetes, traces of glucose will make their appearance. Those with a hereditary tendency of a marked copper dissolving quality of the urine are to be treated like individuals with mild diabetes. MILL.

Rachitis and Internal Secretion—E. ROMINGER, *Zeitschr. f. Kinderheilkunde*, Vol. XI, Nos. 5 and 6, 1914.

Author approached the question whether or not it be possible to demonstrate in rachitis a disturbance of the glands with an internal secretion. For this purpose he employed the dialyzation method of Abderhalden. As regards the thyroid, thymus, ovaries and testicles he obtained absolutely negative results. MILL.

Basedow's Disease and Female Genital Organs—E. v. GRAFF and J. NOVAK, *Archiv f. Gynäkologie*, Vol. CII, No. 1.

Results of the examination of 36 women. Basedow's disease is not rarely associated with anatomical and functional changes of the genitals. There are cases, however, in which the genital area is not at all affected by the presence of the disease. Genital changes may ensue primarily, and may even in a measure compensate for Basedow's disease. However, genital disturbances may occur as a result of an autochthonous Basedow's disease. MILL.

Thyroid Gland and Female Genitals—E. v. GRAFF, *Archiv f. Gynäkologie*, Vol. CII, No. 1.

Enlargement of the thyroid gland during pregnancy is mostly due to the enlargement of an already existing struma. Among all the cases there are only from 9 to 15 per cent. which have ensued without a previously altered gland. Parturition causes frequently, however not invariably, a further enlargement of the thyroid. This is again reduced after parturition. Alimentary glycosuria is very

much intensified in pregnant women with struma. This leaves no doubt that the thyroid exerts a special influence in this regard. On the other hand, albuminuria occurs more often when no enlargement of the gland has ensued. Ingestion of ovarian preparations was without effect upon the thyroid gland. The assumption, according to which struma and myoma are often associated, is an erroneous one. MILL.

Cardiovascular Insufficiency on Thyrotoxic Basis—SCHMIDT, Verein deutsch. Aerzte, Prag, Münchener med. Wochenschr., Nov. 24, 1914.

Demonstration of a case of cardiovascular insufficiency on the basis of a thyrotoxicosis. There existed a swelling of the upper extremities, the chest, back, abdomen and thighs, ascites and bilateral hydrothorax, and some edema of the calves. The right eyelid was abnormally opened, and Graefe's symptom was distinctly positive. Other symptoms on the part of the sympathicus were not present. The thyroid gland was not enlarged. Five years ago similar symptoms existed and the thyroid was enlarged. After 6 months these symptoms subsided. For the following $2\frac{1}{2}$ years the condition was perfectly normal. MILL.

Acquired Disease of the Thyroid—J. ROGERS, Annals Surg., Sept., 1914.

Partial thyroidectomy is to be especially avoided in patients who have not completed or who have just completed their growth and development. Cases of symmetrically enlarged thyroids seem peculiarly prone to relapse even after a considerable period. Operation is to be avoided in subjects of the very nervous type. Patients with marked exophthalmus of long duration bear any operation badly and can seldom be more than moderately improved. Of 62 patients who applied for relief after one or more partial thyroidectomies, only the minority had failed to show any benefit, and a considerable part of this minority stated that they were worse than before the operation. SACHS.

INFECTIOUS DISEASES

Hereditary Syphilis—P. C. JEANS and E. M. BUTLER, Am. Jour. Dis. Child., Nov., 1914.

Authors found that 33 per cent. of the syphilitic children over one year had permanent disabling damage. Eighteen per cent. of such children had long-continued disabling damage. There is about 5 times as much feeble-mindedness in syphilitic families as in non-syphilitic families. The mortality for artificially-fed syphilitic infants, is five times as high as for breast fed syphilitic infants, not including deaths due to intestinal disturbances. SACHS.

Congenital Syphilis—L. FINDLEY and M. E. ROBERTSON, Glasgow Med. Jour., Dec., 1914.

A condition of chronic eczema situated at the angles of the mouth and invading the mucous membranes of the lips is often of a syphilitic nature. Congenital heart disease is not infrequently found in patients suffering with congenital syphilis, as is evidenced by a positive Wassermann reaction. About 45 per cent. of cases of spastic diplegia and 60 per cent. of mental defectives also seem to be the subjects of congenital syphilis as is shown by a positive Wassermann reaction. SACHS.

Tuberculosis, a Disease of Childhood—K. F. ANDWORD, Beiträge z. Klinik d. Tuberkulose, Vol. XXIX, No. 1.

Under the present conditions, the majority of cases of tuberculosis, date back to an infection in childhood. Such early acquired tuberculosis, if it is benign in nature, gives rise to a certain degree of immunity which later explains the rather benign course of tuberculosis at a more advanced age. The source of infection in tuberculosis without symptoms must be ascribed to tuberculous individuals, frequently, however, the infection is caused by tuberculous milk. FRY.

Pulmonary Phthisis of School Children—W. OVEREND, Brit. Med. Jour., Dec. 12, 1914.

The coexistence of an intermittent sudden cough in the absence of cases of pertussis; wasting, pallor, and tiredness; a subfebrile temperature, rising occasionally to 100 deg. F.; the presence of capillary veins and a growth of hair over the upper thoracic spines; and the absence of any obvious disease, should lead to a careful examination of the case for pulmonary tuberculosis and enlarged bronchial glands. SACHS.

Alcoholism and Tuberculosis—HOLITSCHER, Beiträge z. Klinik d. Tuberkulose, Vol. XXIX, No. 2.

Results of the study of 2720 pertaining cases of international origin. The number of alcoholics among the young tuberculous is decidedly less than would correspond to the average population. On the other hand, among the tuberculous of more advanced age a surprisingly large percentage of alcoholics is encountered. FRY.

Subnormal Temperature in Tuberculosis—A. K. STONE, Bost. Med. and Surg. Jour., Dec. 31, 1914.

In pulmonary tuberculosis, usually succeeding the active febrile stage, there is a period in which the temperature is subnormal, that is, it does not rise above 98.6 degrees F. This period of subnormal temperature may last for weeks. Author is of the opinion that the

persistent subnormal temperature is an indication that the case is on the whole progressing favorably. SACHS.

Tuberculous Rheumatism and other Forms of Larved Tuberculosis—

E. NOHL, *Beiträge z. Klinik d. Tuberkulose*, Vol. XXIX, No. 2.

Author mentions among the larved forms of tuberculosis, (1) inactive-latent tuberculosis, which in the absence of clinical symptoms may be recognized by the employment of specific diagnostic means; (2) active-latent tuberculosis, representing a genuine tuberculous affection of an organ, but appearing under different symptoms; (3) tuberculous intoxication. FRY.

Von Pirquet Test—T. FRAZER, *Med. Rec.*, Jan. 9, 1915.

A positive Von Pirquet reaction is less frequent in children than it was once thought to be, the high percentage of reactions obtained being due to the application of the test to the infected children of the poorer classes. Therefore a positive reaction is of greater significance than it is commonly supposed to be. While there is an increasing percentage of reactions with years, and a corresponding decrease in the value of the reaction, the view usually held that the reaction has significance only during the first two or three years of life is not borne out by recent figures. We should be suspicious of a reaction occurring up to the age of ten. A negative reaction, negative on repetition of the test, is valuable evidence of the absence of tuberculosis, unless the child is suffering with advanced or acute disease, especially measles. SACHS.

Eosinophile Sputum Cells, especially in Tuberculosis—WENDENBURG,

Beiträge z. Klinik d. Tuberkulose, Vol. XXIX, No. 1.

Local eosinophilia may be caused by a chronic inflammatory irritation which effects proliferation, transmigration and emigration of the capillary endothelia of the vicinity. In chronic tuberculosis, a peribronchiolytic inflammation induces a suppurative discharge in the bronchial tubes without the presence of bacilli in the expectoration. A suppurative sputum, the suppurative character of which cannot be explained by the presence of specific microorganisms, may with reasonable certainty be characterized as tuberculous when the eosinophiles are present to the extent of 5 per cent. or more. FRY.

Albumin Reaction of Sputum—E. G. GLOVER, *Brit. Jour. Tuberculosis*, Oct., 1914.

Author made 210 quantitative examinations for albumin. The sputum of tuberculous patients contains on the average a larger amount of albumin than that of the non-tuberculous, but a larger amount is sometimes found in the sputum of the non-tuberculous

than in the tuberculous. Injury to the epithelium of any part of the respiratory or buccal tract may give rise to the presence of albumin in the sputum. This reaction is therefore, not of much material assistance in the diagnosis of doubtful cases of pulmonary tuberculosis.

SACHS.

Albumin in the Sputum in Tuberculosis—C. H. COCKE, *Am. Jour. Med. Sci.*, Nov., 1914.

Author concludes that all cases of pulmonary tuberculosis showing bacilli give a positive albumin reaction in the sputum. Frequently albumin will be found in incipient tuberculosis before bacilli are present, but its presence is variable and cannot be relied upon as a definite means of diagnosis in incipient cases. The heavier the albumin content, the greater the activity of the tuberculous process. Fibrosing or proliferating forms of tuberculosis fail to show an albumin reaction in the sputum, also cases cured for two years or more will not show it. When physical signs and the tuberculin reaction fail to show activity, the presence of albumin in the sputum may do so.

SACHS.

Anemia and Infection—H. PRIBRAM, *Deutsches Archiv f. klin. Medizin*, Vol. CXVI, Nos. 5 and 6.

Report of the case of a woman, 39 years old, who was affected with genital hemorrhages for one year when a blood-picture resembling that of myelotic leukemia developed. After an intervening pneumonic infection the blood-picture became soon normal again.

WESTERN.

Pneumococcal Tonsillitis—E. LESCHKE, *Münchener med. Wochenschr.*, Dec. 29, 1914.

Among the various forms of tonsillitis, those caused by a pneumococcal infection play a specific part. They are characterized clinically by their stubborn persistence and marked disturbance of the general health, which is the case even when the local condition is seemingly very mild. Bacteriologically, this type of tonsillitis presents the diplococcus lanceolatus as the predominating pathogenic microorganism. The clinical picture of the affection is either that of follicular tonsillitis with plugs or gray-yellowish to gray-greenish exudate, or it is that of pneumococcal influenza, or finally, that of septic pneumococcal tonsillitis, respectively pneumococcemia.

MILL.

Complement-Fixation in Variola—A. KLEIN, *Münchener med. Wochenschr.*, Nov. 24, 1914.

There is no doubt that in the serum of smallpox patients antibodies can be demonstrated by means of the complement-fixation reaction. The complement-fixation test in variola differs from

serum reaction in syphilis; contrary to the latter it is specific, i. e. there is a genuine antigen-antibody reaction in which in all probability the etiological factors of variola are themselves the antigen. At the present time the only available antigen seems to be the material from the small-pox pustules. All other antigens are unreliable and should therefore not be employed. In order to obtain a greater constancy of this antigen, author recommends the employment of pustule crusts. MILL.

Complement Fixation in Variola—A. v. KONSCHegg, *Münchener med. Wochenschr.*, Jan. 5, 1915.

Author, who has experimentally approached the subject of complement fixation in variola, advances the following conclusions: (1) In the serum of patients affected with variola specific antibodies are present; (2) only such antigens effect complement deviation which are undoubtedly the instigators of variola. MILL.

Staphylococcic Erysipelas—F. REICHE, *Zentralblatt f. innere Medizin*, 1914, No. 44.

There are some cases of erysipelas which are caused by staphylococci. Author reports the clinical history of a case of erysipelas which was typical in every respect, but in which the staphylococcus albus alone was found. The onset of the infection ensued through a superficial lesion on the bridge of the nose. The case terminated lethally. In the heart blood the staphylococcus albus was also demonstrated. In erysipelas due to staphylococcus infection, staphylococcemia with very severe pyemic manifestations occurs frequently. WESTERN.

Simplification of the Diazo Reaction—M. RHEIN, *Feldärztliche Beilage, Münchener med. Wochenschr.*, Dec. 8, 1914.

The impossibility of having the reagents for the Ehrlich diazo reaction always on hand, especially when treating soldiers in a field hospital, prompted author to make use of Weiss' test. The latter is based upon the same principle as the diazo reaction, i. e. the oxidation of urochromogen. The test is performed as follows: The urine in the test tube is diluted until it loses its color, ordinarily, the addition of twice or three times its bulk of water suffices. From 3 to 10 drops of a solution of potassium permanganate (1:1000) are added to the diluted urine. In case the reaction is positive, a distinct gold-yellow coloration will ensue. When negative, either no coloration at all will occur, or but a light brown hue will appear. —Author has employed this test in about 100 cases of typhoid fever. It always corresponded to Ehrlich's diazo reaction. In about 10 per cent. of the latter in which the color of the foam was not distinct, Weiss' test showed a positive diazo reaction. The method is

so simple that author uses it exclusively during daylight. In the presence of artificial light Ehrlich's reaction is more distinct. A further simplification of this test is the direct addition of a small grain of potassium permanganate to the diluted urine. If the diluted urine is at once shaken after the permanganate has been added, a positive reaction will immediately denote itself by a beautiful gold-yellow coloration. In case the test is negative a brownish suspension will ensue after a few seconds. MILL.

Simplification of Gruber's (Widal's) Reaction—L. v. LIEBERMANN and J. ACEL, *Deutsche med. Wochenschr.*, Dec. 10, 1914.

It is not necessary to prepare serum from the blood or to place this into bouillon or physiological sodium chlorid solution, and to remove the blood cells by centrifugation. The test may be materially simplified by collecting the blood in distilled water (2 drops of blood from the tip of the finger in 1 cc. distilled water), and, after the immediately ensuing of complete hemolysis, to employ the colored, clear blood solution for the agglutination test. MILL.

Tonicity of the Abdominal Muscles in Enteric Fever—A. PATRICK, *Quart. Jour. Med. (London)*, Oct., 1914.

Author describes a condition of the abdominal muscles which was first noted by Gardner. It is a slight superficial resistance over a part or the whole of the anterior abdominal wall, with increased superficial resistance in the right hypochondrium. It is recognized by light pressure with fingers. When deeper pressure is made, the resistance is overcome, and it is not recognized as long as this pressure is kept up. When the fingers are run lightly over the surface of the abdomen in these cases, the sensation resembles that produced by running the fingers lightly over soft dough. It is quite different from the deep resistance which is found in cases of general peritonitis, where increase of pressure is met with by increase of resistance. SACHS.

Autopsies of Two Typhoid Carriers—GOEBEL, *Zeitschr. f. Hygiene u. Infektionskrankheiten*, Vol. LXXVIII, No. 3, 1914.

The necropsy of two chronic typhoid bacilli carriers showed typhoid bacilli in the bile of both. The bacilli were also demonstrated in gall-stones found in one of the cases. MILL.

Diagnosis of Osteomyelitis—M. B. CLOPTON, *Surg., Gynecol. and Obstet.*, Jan., 1915.

Emphasis is laid on the fact that the earliest symptom of osteomyelitis is pain in the shaft of the long bone (usually near the end), accompanying septic symptoms. In a few cases of profound sepsis, the pain-sense is lost. Swelling of the shaft near the joint,

frequently at the epiphyseal line, occurs early. In septic arthritis, there is usually less pain and several joints are involved. Tuberculosis is a chronic infection and involves the epiphysis. In the later stage of acute osteomyelitis, the Röntgen ray is of great value.
SACHS.

RESPIRATORY AND CIRCULATORY ORGANS.

Epistaxis in Later Childhood—H. PETRY, Berliner klin. Wochenschr., Dec. 7, 1914.

Local inspection of the nose is imperative whenever there ensues epistaxis in children. This is also necessary when no fever is present. Although in most of the instances nothing but a simple ulceration of the septum will thus be revealed, occasionally a chronic nasal diphtheria may be encountered. This is particularly the case in the nursing. In sepsis and affections resembling it as well as in all infectious diseases, the diagnosis "septic epistaxis" is not permissible without a scrutinizing examination of the nose. Hemorrhage due to a simple ulcer of the septum may aggravate the course of the disease and thus call forth the clinical picture of grave septic infection.
MILL.

Percussion of the Lungs—N. K. WOOD, Med. Press (Lond.), Dec. 23, 1914.

The present methods of percussion are of little value, because, far from having any universal standard of dulness, there is not in most instances, even an individual standard. Upward percussion is an absolute essential of correct work. The old method of downward percussion should be discarded, and no longer taught to medical students. Wood adopts the following standard which he says is definite and easily learned. This standard can be readily acquired by the ear, and by the musical sense of both hands, measured by the force of the plexor blow and the resistance under the pleximeter. Normal resonance is F to F sharp below middle C; 1-line dulness or impaired resonance is A below middle C to middle C; 2-line dulness or definite dulness is E flat to F above middle C; 3-line dulness or very marked dulness is B flat below immediate C; 4-line dulness or flatness is E to F above intermediate C.

SACHS.

Syphilis of the Lungs—E. A. BURHAM, Boston Med. and Surg. Jour., Sept. 10, 1914.

In its early stages, syphilis produces pulmonary conditions like bronchitis and bronchopneumonia, but it most frequently affects the lungs in the tertiary stage, when its lesions produce physical signs which are identical with those of pulmonary tuberculosis. The patient with pulmonary syphilis does not appear to be so ill as one

with a tuberculous infection of the same extent. Hemoptysis is more frequent in tuberculosis, the temperature curve is more variable than in syphilis, while the latter condition does not produce so rapid a loss of flesh and strength. The absence of bacilli in the sputum together with the presence of any of the stigmata of syphilis, and especially with a positive Wassermann reaction should make a diagnosis certain.

SACHS.

Cases of Lung Abscess—C. L. SCUDDER, Bost. Med. and Surg. Jour., Oct. 1, 1914.

Lung abscess may follow an embolism after an ordinary surgical operation. Sudden severe pain in the chest, associated with a rise of temperature and pulmonary signs following surgical operations should suggest the possible beginning of a lung abscess. Many intraabdominal infections are associated directly with an infection through the diaphragm of the diaphragmatic pleura, and then of the lung with lung abscess. The Röntgen ray is of great value in the diagnosis of this condition.

SACHS.

Primary New Growths of the Mediastinum—J. N. W. ROSS, Edinburgh Med. Jour., Dec., 1914.

Malignant disease of the mediastinum is not so rare as is generally supposed, whereas innocent tumors of the mediastinum are exceedingly rare in England. It is essentially a disease of early middle life, but it may occur at any age. It seems to be increasing in frequency. The disease is twice as common in males as in females. A definite tuberculous history is often present in these cases. The anterior mediastinum is the common seat of origin and lymphosarcoma is the commonest form of malignant disease. Carcinoma of the mediastinum is more frequent in females than in males. The lungs are practically always affected sooner or later, and pulmonary symptoms are generally present. Extra-thoracic metastases occur frequently. Hemoptysis though moderately common is rarely severe. A pleural effusion is often present and it is frequently hemorrhagic, but a hemorrhagic effusion is not pathognomonic of malignant disease of the mediastinum.

SACHS.

Normal Peculiarities of Heart Sounds in the Region of the Sternum—G. BLUMER, Arch. Int. Med., Oct., 1914.

Author describes the normal cardiac sound underneath the sternum as a superficial scratching sound. It occurs at all ages after infancy. It is heard throughout both systole and diastole and is of brief duration. The sound appears to be superficial, strongly suggests a friction rub, but does not resemble an endocardial murmur. It is uninfluenced by respiration. It is usually heard best at the lower end of the sternum opposite the fifth and sixth costal

cartilage, a little to the left of the medium line. This sound is a physiological phenomenon, and the author is confident that it will be an occasional source of error. SACHS.

Secondary Sounds over the Aorta—KÜLS, *Zeitschr. f. klin. Medizin*, Vol. LXXX, Nos. 5 and 6.

Report of 31 observations of systolic sounds which were localized in the second intercostal space. The character of these sounds was uneven, scraping and rough. In 12 instances accentuation of the second aortic sound and extension of the aorta in the Röntgen shadow were found; enlargement of the heart toward the left was demonstrated in 17 cases. Blood pressure was above 160 mm. Hg. in 7 cases, between 140 and 160 mm. Hg. in 13 cases, and below 140 mm. Hg. in 11 cases. Subjective symptoms occurred in but 10 cases; in 5 cases angina pectoris was present. In 16 cases the sounds were of an adventitious secondary nature, and were met with in a purely accidental manner. Polyarthritis and syphilis could be excluded in 17 of the cases. Artherosclerosis plays probably the principal part in the production of these secondary sounds. According to the clinical manifestations, a typical stenosis of the aorta did not exist in any of these cases. The sound is always more or less adventitious and must be caused by a roughened aortic wall or by changes of tension in the vessel. WESTERN.

Blood and Pulse Pressure in Arteriosclerosis—W. JANOWSKI, *Zeitschr. f. klin. Medizin*, Vol. LXXX, Nos. 5 and 6.

In 24 out of 200 cases of arteriosclerosis the blood pressure was increased. In 88 cases the blood pressure varied between 120 and 160 mm. Hg., in 51 cases between 180 and 200 mm. Hg.; in 6 cases the pressure was above 200 mm. Hg. Of 62 sclerotics who had survived myocardiac decompensation, 42 exhibited normal blood pressure; only 4 had a pressure between 140 and 160 mm. Hg. The low pressure is caused by the far-advanced heart affection. Pulse pressure of 21 of 42 patients thus affected was between 30 and 40 mm. Hg. Of 18 patients with increased blood pressure who had had cardiac decompensation, but 3 presented a lower pulse pressure than 60 mm. Hg. In 50 cases of arteriosclerosis with renal involvement myocarditis existed positively in 4 cases. In these the blood pressure varied between 128 and 144 mm. Hg., the pulse pressure between 48 and 90 mm. Hg. In the other cases the blood pressure was considerably increased, in 29 above 180 mm. Hg. The pulse pressure in 75 per cent. of patients of this group was between 70 and 135 mm. Hg. The cases of arteriosclerosis associated with nephritis showed the highest blood pressure. In 7 cases of acute nephritis the blood pressure was between 130 and 185 mm. Hg. In 16 cases of chronic nephritis the blood pressure and pulse pressure were normal; in 84 they were increased. WESTERN.

Electro-Cardiography—J. F. H. DALLY, *West London Med. Jour.*, Oct., 1914.

Author is of the opinion that one of the chief fields of utility of the electro-cardiograph in practice is the ease with which one can pick out and classify the functional disorders which are oft-times a source of such great discomfort to the patient, and thus we are able to state with much greater confidence an opinion as to prognosis. Many of the hearts condemned in former years because of symptoms which cause anxiety and pain, when treated as a result of the knowledge gained from this and other modern cardiac research, will prove to have still many years of useful activity.

SACHS.

Heart-Block in Acute Rheumatic Carditis—A. E. NAISH and A. M. KENNEDY, *Lancet*, Nov. 28, 1914.

Two cases of acute rheumatic carditis in children are reported by authors in which heart-block occurred. Both cases were associated with similar histological changes in the cardiac musculature.

SACHS.

Pulsus Paradoxus—A. W. FALCONER and J. M. McQUEEN, *Quart. Jour. Med.* (London), Oct., 1914.

Authors state that in addition to the ordinary sinus arrhythmia, at least two entirely different conditions have been included under the term pulsus paradoxus: (1) Cases presenting complete obliteration of the pulse during deep inspiration, and due in their opinion to oval deformation of the subclavian artery in the subclavian triangle; (2) Cases occurring in a great variety of conditions, in which the one common factor is an embarrassment of the circulation, and in which the normal inspiratory diminution of the blood pressure is a sufficient explanation.

SACHS.

Production of Gallop-Rhythm—M. GUBERGITZ, *Deutsches Archiv f. klin. Medizin*, Vol. CXVI, Nos. 5 and 6.

Gallop-rhythm is observed in various forms of myocardial weakness, especially in interstitial nephritis and in certain infectious diseases. It is mostly associated with hypertension. When gallop-rhythm is associated with low blood pressure, the prognosis is less favorable. The mechanism of the production of the various forms of gallop-rhythm (presystolic and protodiastolic gallop) is an entirely different one as is evinced by the electrocardiogram.

WESTERN.

Clinical Symptoms of Beginning Cardiac Weakness—MORITZ, *Münchener med. Wochenschr.*, Jan. 5, 1915.

A clinically important form of dyspnea is the nocturnal cardiac asthma. The patients may be troubled for a long period, occasionally almost every night, with transitory dyspnea which rouse

them out of sleep and compel them to sit up. During the day no especially marked difficulties may arise. It is even possible that the patient is able to take a comparatively long walk. Frequently the nightly attacks of dyspnea and oppression are associated with a good deal of irritation to cough. This cough may once in a while remove a somewhat tenaceous sputum. As soon as this expectoration has taken place the patient will breathe easier. Such cases may be mistaken for instances of bronchial asthma. It is rather suspicious when during a nocturnal cough seizure an abundant, thin and foamy expectoration is eliminated. When this is the case, then one is most always confronted with a mild attack of pulmonary edema. A form of cardiac asthma that is by no means rare is the frightened startling with a sense of oppression at the moment of falling asleep. The patient, though extremely sleepy, may be kept from falling asleep by this phenomenon, which is apt to recur frequently. These nocturnal symptoms occur comparatively often in patients with high blood pressure. Many of these, as is well known, have contracted kidney. During the day, the pulse, as far as fulness and frequency are concerned, may be within normal limits.

MILL.

Symptomatology of Abdominal Angina—M. J. BREITMANN, *Zentralblatt f. innere Medizin*, 1914, No. 46.

Abdominal angina is caused by sclerosis of the abdominal blood vessels or by vascular spasm. It has the same etiology as atherosclerosis in general. Among the symptoms are, painful meteorism due to intestinal paresis; pains, often very violent in character; epigastric pulsation; pallor; vertigo; syncope, and pressure sensitiveness of the aorta. The symptoms occur periodically. The diagnosis must necessarily exclude other diseases of the abdominal organs. The prognosis is unfavorable, as a general rule, as the symptoms are often an expression of the beginning of the end.

WESTERN.

ALIMENTARY TRACT

Prognosis in Cancer of the Tongue—W. TROTTER, *Lancet*, Oct. 24, 1914.

A favorable prognosis as to the danger of an operation for cancer of the tongue can always be given in cases of edentulous patients. If a man develops cancer of the mouth or pharynx, the possession of natural teeth is a misfortune almost as great as the malignant disease itself. The precocious development of early diffuse invasion seems particularly liable to occur in strong, healthy, well-nourished subjects of florid appearance and comparative early age. A glandular enlargement in which the glands are ill-defined and rather soft so that they are not always distinctly palpable offer a very bad prognosis.

SACHS.

Esophagitis Dissecans Superficialis—E. KOECHLIN, *Korrespondenzblatt f. Schweizer Aerzte*, No. 35, 1914.

The patient was a woman, 35 years old. She was affected with the rare disease described as esophagitis dissecans superficialis. The woman recovered after vomiting membranes of which one was 25 cm. long.

MILL.

Volatile Fatty Acids in Fresh and Spoiled Milk, and the Pathogenesis of Digestive Disturbances in the Nursing—H. BAHRDT and F. EDELSTEIN, *Zeitschr. f. Kinderheilkunde*, Vol. XI, Nos. 5 and 6, 1914.

Author made some experiments with fresh milk which was kept at room temperature for 2 days and with milk that had been impregnated with pure cultures of various bacteria and had also been left standing for some time. In neither of the specimens of milk could an amount of volatile fatty acids be demonstrated that was sufficiently large to give cause to digestive disturbances, especially to summer diarrheas.

MILL.

Psychogenous Anomalies of the Gastric Secretion in Childhood—W. WEGENER, *Archiv f. Kinderheilkunde*, Vol. LXIII, Nos. 5 and 6.

Among 80 children with disturbances of the stomach, 19 presented pathological composition of the gastric juice, that is, 2 had hyperacidity and 17 pronounced anachlorhydria. Such disturbances occur comparatively frequently in older children. A neuropathic constitution is most always underlying such secretory anomalies. The children affected with these secretory disturbances are mostly more or less undernourished, are pale and exhibit some degree of vasomotor paresis; their conjunctival and tracheal reflexes are either missing or markedly diminished; the deep reflexes are increased and there is a mechanical overexcitability of the peripheral nerves and positive Rosenbach phenomenon. Occasionally a relationship with the exudative diathesis seems to exist. The prognosis of these nervous disturbances in childhood is favorable.

MILL.

Gastrogenous Diarrheas and the Occurrence of Achylia Pancreatica in Achylia Gastrica—A. BITTORF, *Deutsche med. Wochenschr.*, Nov. 5, 1914.

Severe creatorrhea and mild steatorrhea ensue frequently in diarrheas due to achylia gastrica. The amount of trypsin in the stools and the stomach is nevertheless normal in many of these cases. The abnormal state of the feces does not prove the presence of a functional achylia pancreatica. The latter ensues infrequently; the diminution of trypsin in achylia occurs somewhat oftener. The unequal utilization of the ingesta in achylia is the consequence of increased peristalsis and insufficient gastric digestion. The examination by means of the Röntgen rays showed in these diarrheas a

moderately increased evacuation of the stomach and small intestine, and more rapid emptying of the colon. The latter phenomenon is frequently the result of a catarrhal condition that has ensued on the basis of a chemical or bacterial alteration. MILL.

Perforated Gastric Ulcer—A Collective Report, Edinburgh Med. Jour., Dec., 1914.

In the vast majority of cases the patient suffered with indigestion of a severe type for a long period before perforation occurred. Hematemesis was much more common in women than in men. No information could be elicited in the records of gastric perforations to indicate that the occurrence of perforation is heralded by any characteristic symptoms. In severe cases, the indigestion had been distinctly worse than usual for some days before. In practically every case the initial symptom was agonizing pain in the epigastrium coming on with great suddenness and severity. In the great majority of cases the pain was situated in the epigastrium and, as a rule, towards the left of the middle line. Vomiting is a much more common symptom in perforation of the stomach than in duodenal perforation. In most cases the main tenderness was over the epigastric and left hypochondriac regions, and this was found to correspond fairly accurately with the site of the perforation. By the time the patient comes under observation, general muscular rigidity is as a rule present. Diminution or loss of liver dullness was recognized in 127 of the 175 cases in which it was noted.

SACHS.

Acute Gastric and Duodenal Perforating Ulcer—F. FEE, Lancet-Clinic, Jan. 16, 1915.

Acute gastric and duodenal ulcer must be differentiated in the early stages from acute thoracic lesions, but no great difficulty should exist here if we remember to make a complete physical examination of the patient, when pleurisy or pneumonia will be revealed by the usual signs. Perforation of the gall-bladder is one of the most difficult conditions to differentiate, for the symptoms and signs may be almost identical. In such cases one must rely upon the previous history. Acute pancreatitis in a patient with a very thick abdominal wall may present many difficulties in differential diagnosis. In these cases the pain is of a different character, and is not limited to the right side as in duodenal perforation. Vomiting is more frequent. The pulse in early perforation is not increased in frequency, but is weak, rapid and thready in acute pancreatitis. In ordinary cases the swollen gland can be easily palpated. The characteristic rapid, jerky respiration also is absent in acute pancreatitis.

SACHS.

Boas-Oppler Bacillus—H. M. GAULT and C. C. ILES, *Jour. Path. and Bact.* (London), Oct., 1914.

A comparison of the Boas-Oppler bacillus with the *Bacillus Bulgaricus* tends to prove that it is identical with the latter organism and not an organism *sui generis*. Authors believe that in cases of cancer of the stomach it is the absence of hydrochloric acid that allows of the growth of the organism, and that the lactic acid is formed as a result of the activity of this bacillus. SACHS.

Duodenal Ulcers in Infancy—B. S. VEEDER, *Am. Jour. Med. Sci.*, Nov., 1914.

Author reports 5 cases of duodenal ulcer in infants. In 4 of the cases a clinical diagnosis was made which was later confirmed in 3 instances at autopsy, and in one case the lesion was found post-mortem. In the fourth case the infant recovered. All the infants were under 6 months of age and marasmic. In none of the cases had there been an acute gastrointestinal disturbance. Vomiting was present in all the cases. The presence of gross hemorrhage from the bowel is the only definite symptom pointing to a duodenal ulcer, and hence when this symptom, which may be looked upon as a complication, is absent, the diagnosis cannot be made. It is necessary to exclude ulcerative lesions of the lower intestine, anal fissure, etc. SACHS.

Dysentery-like Affections of the Bowels—E. LESCHKE, *Deutsche med. Wochenschr.*, Dec. 3, 1914.

Observations in 8 cases (soldiers). There are intestinal affections with bloody and slimy discharges which exactly resemble genuine bacillary dysentery, but which, etiologically, differ very much from it. They are caused by other enterogenous infections or by parasites, or by enterogenous intoxications; they may also be due to parenteral infections, as through the tonsils, or to mechanical damage of the intestinal wall. Anaphylactic enteritis may also resemble dysentery. MILL.

Insufficiency of the Ileocecal Valve in the Röntgen Picture—E. MARCUSE, *Berliner klin. Wochenschr.*, Dec. 21, 1914.

The insufficiency of the ileocecal valve as determined by the röntgenogram is an interesting incidental discovery which does not permit of any diagnostic conclusions. MILL.

Acute Appendicitis and Acute Appendicular Obstruction—D. P. D. WILKIE, *Brit. Med. Jour.*, Dec. 5, 1914.

Two acute pathological processes are met with in the vermiform appendix, i. e., acute appendicitis and acute appendicular obstruction. Clinically, acute appendicitis is distinguished by the signs of inflammation, there being from the onset a rise in temperature

and pulse. Acute appendicular obstruction gives rise to vomiting, colicky pain and abdominal tenderness, but at the onset to no appreciable rise in pulse or temperature. The change occurring in an appendix, the lumen of which is completely obstructed, depends on the presence or absence of fecal matter within its lumen.

SACHS.

Pruritis Ani—L. E. C. NORDBURY, Practitioner (London), Nov., 1914.

A thorough investigation of the rectum and pelvic colon by means of the sigmoidoscope should be carried out in every case of pruritus ani in which an external examination or an examination of the anal canal does not reveal a satisfactory cause. Pruritus ani may be caused by an irritating discharge from simple or malignant growths in the bowel, from certain forms of colitis or proctitis; granular proctitis or the hypertrophic catarrhal variety. SACHS.

Fibrinolysis in Chronic Hepatic Insufficiency—E. W. GOODPASTURE, Johns Hopkins Hospital Bull., Nov., 1914.

Specimens of blood from 4 cases of atrophic hepatic cirrhosis have possessed the property of completely digesting their clot within a few hours, at body temperature. Normal blood will not digest its clot for days or weeks. Dissolution of clot in the blood of cirrhosis cases is due to an enzyme. Its activity is destroyed by heat; inhibited by normal serum; and diminished in old plasma. Fibrinogen content of the blood of each case has been below normal. Hydræmia was a constant feature. One case exhibited a low phenoltetrachlorphthalein output. Digestion of clots in vivo explains in part the hemorrhagic tendency present in these patients. WESTERN.

Hepatic Functional Tests (Galactose and Phenoltetrachlorphthalein)—W. R. Sisson, Arch. Int. Med., Dec. 15, 1914.

Forty grams of galactose are given by mouth at 6 A. M. in 200 or 300 c.c. of tea. The usual breakfast is given one hour later. The urine is collected for a period of 6 hours after the ingestion of the galactose, at 2 hour intervals, and tested for sugar. In the majority of cases of normal individuals, less than one gram is excreted. Patients with icterus catarrhalis have an intolerance to galactose. The galactose test may be used in differentiating icterus catarrhalis from other hepatic conditions showing biliary stasis. It is rarely positive in cases of cirrhosis of the liver showing no evidence of biliary stasis. Phenoltetrachlorphthalein when given subcutaneously to a patient escapes only in the bile and the feces are tested for it. The normal output is about 35 per cent. Further studies must be undertaken before any definite conclusions can be drawn concerning the value of this test. It offers a means of diagnosing certain cases of cirrhosis of the liver and of neoplasms

of this origin. (See Archives of Diagnosis, Vol. VI, p. 394, and Vol. VII, p. 193.) SACHS.

Ultimate Results in 160 Gall-Stone Cases—J. G. CLARK, Am. Jour. Med. Sci., Nov., 1914.

The worst results occur among the combined cases in which there are manifest symptoms due to biliary changes. The ratio of cures is in direct proportion to the severity of the symptoms. The weight of evidence most emphatically favors the early removal of gall-stones whether they produce symptoms or not. SACHS.

NERVOUS SYSTEM

Lange Gold Chlorid Reaction—C. G. GRULEE and A. M. MOODY, Am. Jour. Dis. Child., Jan., 1915.

In the cases of congenital syphilis included in this report, the cerebrospinal fluid reacted to the colloidal gold solution always in lower dilutions, and with a marked degree of regularity, strongest in the dilutions of 1:40 and 1:80. There is a small group of cases of congenital syphilis in which the reaction is similar to that of parietic dementia. The reaction as obtained in congenital syphilis is most nearly approached by those conditions which show a slight inflammation of the meninges or brain, and are not likely to be confused clinically with syphilis. The reaction in tuberculous meningitis is found to be most intense in the dilutions of 1:160 and 1:320. It is evident that the Lange gold chlorid reaction is of value only as an aid in diagnosis. SACHS.

Chronic Subdural Hemorrhage of Traumatic Origin—W. TROTTER, Brit. Jour. Surg., Oct., 1914.

Internal hemorrhagic pachymeningitis is a term which should be discarded in favor of some such term as chronic subdural hemorrhage. Apart from certain cases occurring in diseases which have a strong tendency to spontaneous hemorrhage, and possibly including them, hemorrhagic pachymeningitis is almost if not quite invariably a true traumatic hemorrhage coming from veins in their course between the brain and dural sinus. This condition should be dealt with surgically, with the expectation of a successful immediate and remote result, if the treatment is carried out early. It constitutes a clinical type of disease well marked and characteristic. SACHS.

Glycyl-Tryptophan Reaction in Meningitis—R. H. MAJOR and E. NOBLE, Arch. Int. Med., Sept., 1914.

The glycyl-tryptophan test is a valuable addition to our diagnostic measures in meningitis. Meningitis is consistently present in cases in which the peptolytic index is higher than one. SACHS.

Meningitis in Diphtheria—F. REICHE, *Zeitschr. f. Kinderheilkunde*, Nos. 5 and 6, 1914.

The association of diphtheria with meningitis of most varying bacterial origin is very rare. In 8000 cases of diphtheria the combination ensued but 8 times. The cases which recovered presented the clinical picture of serious meningitis. MILL.

New Physical Sign in Lumbar Lesions—L. W. ELY, *Am. Jour. Orthop. Surg.*, Oct., 1914.

When lesions, either traumatic or inflammatory, of the lumbar spine are present, author states that if the patient be laid on his face and his knee flexed, his pelvis on the side of the lesion will rise from the table. By means of this sign lumbar lesions may be differentiated from those of the sacroiliac joint. SACHS.

Nervous Cretinism—R. McCARRISON, *Brit. Jour. Children's Dis.*, Dec., 1914.

Author is well aware of the objections to the use of the term "nervous cretinism," but in so distinguishing these cases by a distinctive title, he hopes to focus attention on a condition which depends on congenital hypothyroidism. The symptoms are, in brief, a combination of congenital myxedema with congenital cerebral diplegia, in all their varying grades. The signs of derangement of the central nervous system may vary from the slightest degrees of paraplegia to the most intense grades of spasticity, athetosis, fits, and idiocy. Such extreme examples of this type of cretinism may be indistinguishable from cases of cerebral diplegia, and it is only by the recognition of the scanty myxedematous signs of the malady, and by the application of the therapeutic test of thyroid medication that their true nature can be appreciated. SACHS.

Analysis of a Case of Psychasthenia—H. FLOURNOY, *Johns Hopkins Hospital Bull.*, Nov., 1914.

Report of a case of psychasthenia with the following striking points: The sudden onset of an obsessive fear that the patient would kill his child, then that he would kill himself, against which the patient fought in vain for 7 months. The disappearance of the obsession as soon as the patient saw that it could be connected with previous difficulties. The persistence of an uneasy feeling towards the children, without fear, arising at times when the patient is depressed or crossed. The whole trouble is closely related to a suspicious state of mind, a fact which the patient had to realize. His prospect of avoiding a relapse of the obsessive fears depends greatly on the extent to which he will be able to assume a new mental attitude towards his wife. (The case report should be studied in the original.) WESTERN.

Status Lymphaticus in Dementia Praecox—H. EMERSON, Arch. Int. Med., Dec., 1914.

Status lymphaticus in males is characterized by decided scantiness of the hair on chin and upper lip, scanty axillary and sternal hair, scanty or feminine distribution of pubic hair, a slender thorax, rounded contour of upper arms and thighs, with an arching of the latter, hypoplastic external genitals, particularly if associated with cryptorchidism and a delicate velvety skin. The diagnosis is further confirmed if we find hyperplasia of the lymph tissue of nose, throat, and tongue and an increase in the palpable cervical and axillary lymph nodes. The incidence of status lymphaticus in cases of acute alienation in the first three decades is higher than among alcoholics and narcotic drug habitués, in whom there is an incidence of 22 per cent.

SACHS.

Epilepsy and Cerebral Tumor—W. A. TURNER, Brit. Med. Jour., Dec. 5, 1914.

Tumors involving the cortex and subcortical white matter of a cerebral hemisphere may give rise to seizures having features characteristic of idiopathic epilepsy. These attacks may precede the onset of the symptoms and signs of intracranial tumor by many years and render the diagnosis of the true cause of the attack well-nigh impossible. The existence of certain signs, however, favors the presence of an organic lesion; such are a well-defined local warning, the presence of some degree of post-convulsive hemiplegia, inequality of the deep reflexes on the two sides, unilateral abolition or impairment of the abdominal reflexes, and above all the development of an extensor plantar response. It is, therefore, important in all cases of epilepsy to examine the reflexes, and the optic discs from time to time.

SACHS.

URINARY ORGANS—MALE GENITALIA

Seminal Vesiculitis—B. A. THOMAS and H. K. PANCOAST, Annals Surg., Sept., 1914.

Seminal vesiculitis is more prevalent than it is supposed to be. It has a manifold symptomatology, often expressing itself remote from the urinary tract. The disease is analogous to pustules in the female.

SACHS.

FEMALE ORGANS OF GENERATION—PREGNANCY— PARTURITION—INFANTS

Experimental Research concerning Renal Changes in Pregnancy—J. and S. BONDI, Archiv f. Gynäkologie, Vol. CII, No. 1.

The kidney of the pregnant animal is more sensitive to poisons than that of the non-pregnant. Epithelial poisons like uranium



and chromium show especially marked alterations, while arsenic and cantharidin, causing vascular poisoning, give rise to but slight changes. The sensitiveness of the kidney of the gravid animal seems, therefore, to be limited to the epithelium of the urinary tubules, and especially to the convoluted tubules. MILL.

The Kidneys and Heart in Pregnancy—V. J. McALLISTER, Med. Press (London), Nov. 25, 1914.

Heart lesions or kidney lesions complicating pregnancy seldom prove fatal. Occurring together, their association with pregnancy is usually extremely serious in its consequences. Pregnancy determines an increased cardiac activity and the heart slowly hypertrophies to withstand the sudden strain of parturition. Experience shows that the gravest cardiac lesions in this connection are those affecting the cardiac musculature. Where the kidneys are diseased, the behavior of the blood pressure is of great importance. An elevated blood pressure increases greatly the work of the heart.

SACHS.

Backache—C. OGILVIE, N. Y. Med. Jour., Dec. 5, 1914.

Backache is often caused by a postural deformity which produces a muscular strain. Weak feet are a frequent cause of this postural deformity. Sacroiliac joint strain is also responsible for a number of cases. Myalgia is often of an infectious origin.

SACHS.

THE EDITOR HAS RECEIVED A NUMBER OF RECENT PUBLICATIONS WHICH WILL BE REVIEWED, AS FAR AS SPACE PERMITS, IN THE APRIL ISSUE OF THE ARCHIVES OF DIAGNOSIS.

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THE ARCHIVES OF DIAGNOSIS

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AND THE PROGRESS OF DIAGNOSIS AND PROGNOSIS

Vol. VIII

APRIL, 1915

No. 2

FOUNDED AND EDITED BY
HEINRICH STERN, M.D., LL.D.
New York



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Special Articles

THE PATHOLOGY AND DIAGNOSIS OF SO-CALLED
DIABETIC GANGRENE

By LEO BUERGER

Associate Attending Surgeon and Associate in Surgical Pathology, Mt. Sinai
Hospital; Visiting Surgeon, Har Moriah Hospital; Instructor in
Clinical Surgery, Columbia University

New York

The widespread impression that the vasomotor affections of the extremities are related in some way to the gangrene of organic vascular disease, has convinced us that neither the pathological nor the clinical aspect of these maladies is clearly understood. In an extensive pathological investigation of the vascular lesions in forty-five amputated lower extremities taken from cases of thrombo-angiitis obliterans,* and of some thirty amputated lower extremities obtained from cases of arteriosclerotic and diabetic gangrene, a large number of pathologic data were acquired which have enabled us to crystallize and classify our conceptions as to the lesions that attend the different types of gangrene of the extremities. In our series, there was also material obtained from the upper extremities, including an arm amputated for thrombo-angiitis obliterans, and several fingers; and besides this, two gangrenous limbs

*Studies made in the Department of Surgical Pathology, Mt. Sinai Hospital.

in which it could be clearly demonstrated that *gangrene can occur without any organic arterial disease*.

The clinical material at our disposal was still larger, including some two hundred and fifty cases of thrombo-angiitis obliterans and many cases of gangrene of the neurogenic or vasomotor type (Raynaud's disease, chronic acro-asphyxia, etc.), cases of atherosclerotic, so-called diabetic gangrene, and thrombotic and embolic gangrene due to other causes.

Both our pathological and clinical material, therefore, was adequate to allow us to come to very definite conclusions regarding the arterial changes that lead to so-called diabetic gangrene. Current conceptions are so hazy as to the causation of diabetic gangrene, that it may not be amiss to give here a brief outline of the pathological lesions that have been found responsible for this condition. We may anticipate by saying that diabetic gangrene is, in truth, a process of mortification directly referable to extensive arterial disease, the arteries of the affected extremities being intensely atherosclerotic, often occluded over a large part of their course, usually by obturating atheromatous and calcific masses, less commonly by virtue of secondary thrombosis.

For an elucidation of the pathology and diagnosis of diabetic gangrene, we will give a brief summary of the usual lesions encountered in the vessels in these cases, will compare them with the lesions of arteriosclerotic or senile gangrene, and finally give a resumé of the pathology of the vessels in thrombo-angiitis obliterans, since this is the largest class of cases which may offer difficulties in diagnosis. As for the clinical diagnosis, this shall be discussed by giving a summary of some of the more important clinical types of diabetic gangrene, and by differentiating these from thrombo-angiitis obliterans, embolic and thrombotic gangrene, arteriosclerotic gangrene, and gangrene due to vasomotor disturbances.

For purposes of differential diagnosis, we have found the following classification of the vasomotor and trophic disturbances of the lower extremities of particular value. We distinguish trophic disturbances and gangrene according to whether the vessels of the affected region show no organic abnormalities, or as to whether distinct, degenerative, inflammatory or occlusive lesions occur. Thus, we have, first, a *neurogenic variety* including Raynaud's disease,

scleroderma, multiple circumscribed gangrene, erythromelalgia, sclerodactyly and acro-asphyxia or acro-cyanosis; and, second the group in which the arteries show organic lesions either thrombo-angiitis obliterans, arteriosclerosis, and rare lesions of endarteritis and embolic or thrombotic occlusion.

THE PATHOLOGY OF SO-CALLED DIABETIC GANGRENE

A study of the condition of the arteries and veins in limbs amputated for so-called diabetic gangrene, reveals the fact that in each and every instance we are dealing, not with a gangrenous process due to the diabetes *per se*, but a mortifying process dependent upon extensive arterial disease. If we dissect out the larger vessels, including the femoral, popliteal, posterior tibial, peroneal, anterior tibial and plantars with their larger branches, we soon learn this fact, that there is an extensive and intense athero- or arteriosclerotic process. In some cases there is *marked occlusion* due to the heaping up of atheromatous and calcific material, or to a combination of this process and secondary thrombosis, or a moderate degree of atherosclerosis with obturating thrombosis. These cases may be grouped under the caption "intense, obturating, atherosclerotic process." In another series of cases, we find that, although very few of the vessels are completely closed, the atherosclerotic process is very extensive and intense, making the vascular walls rigid, or producing a dilatation or pouching of the walls of many vessels. Both of these lesions will have as their sequence impaired nutrition of the parts, by virtue of the loss of elasticity in the arterial walls. This type may be grouped under the caption "atherosclerosis with slight or no occlusion."

Common to both types, however, is the fact that the larger veins do not share in the atherosclerotic process, although they may at times seem to have suffered a moderate degree of endarteritis or thickening of the intima. In short, *characteristic for so-called diabetic gangrene is the presence of the typical lesions of athero- or arteriosclerosis*. These differ in no way from the lesions of the arteries in arteriosclerotic or senile gangrene, and justify the conclusion that in diabetic gangrene we are dealing with an atherosclerotic or arteriosclerotic process.

The arterial lesions may be summed up as follows: Extensive degeneration of the arterial walls, intense atherosclerosis, calcification, sometimes bone formation, often occlusion of a large part of a vessel's course, the arteries being converted into rigid pipe stems; at other times, less pronounced atherosclerosis with dilatation of the vessel walls in places, and in still other cases a combination of intense atherosclerosis with thrombosis. A reference to Fig. 1* will show the nature of the occlusive process in some of the cases of diabetic and arteriosclerotic gangrene. The elastic tissue stains show a proliferation and heaping up of the elastic layers or lamellae, and that the remnant of the lumen may be occluded by organized clot. Another type of lesion not depicted here, is that in which marked calcification of the vessel walls takes place, sometimes attended with bone formation.

The following abstract from some of my notes on the pathology of the vessels in amputated legs of cases of so-called diabetic gangrene will illustrate in detail the type of lesions that are found.

P. A., April 1, 1909 (diabetic gangrene): Right leg ablated at the knee joint; one ulcer situated at the outer border of the foot, corresponding to the head of the fifth metatarsal; it is about the size of a dime, covered with sluggish, necrotic granulations; a second ulcer, slightly larger, more superficial, found at the head of the first metatarsal, at the inner border of the foot.

Popliteal artery is atheromatous at the point of ablation, possibly thickened but not occluded.

Posterior tibial is almost completely closed by atheromatous plaques. Its middle third is almost completely closed by degenerate atheromatous masses; the lower third shows similar lesions.

External plantar is almost completely closed by atheroma.

Peroneal—in its upper part it is almost completely closed by a series of atheromatous plaques; throughout the remainder of its course, there are yellowish, rounded atheromatous plaques and diffuse atheroma.

Anterior tibial is practically closed by the atheromatous process and calcification throughout its entire extent.

*Photomicrographs were kindly prepared by Dr. F. S. Mandelbaum, Pathologist, Mt. Sinai Hospital.

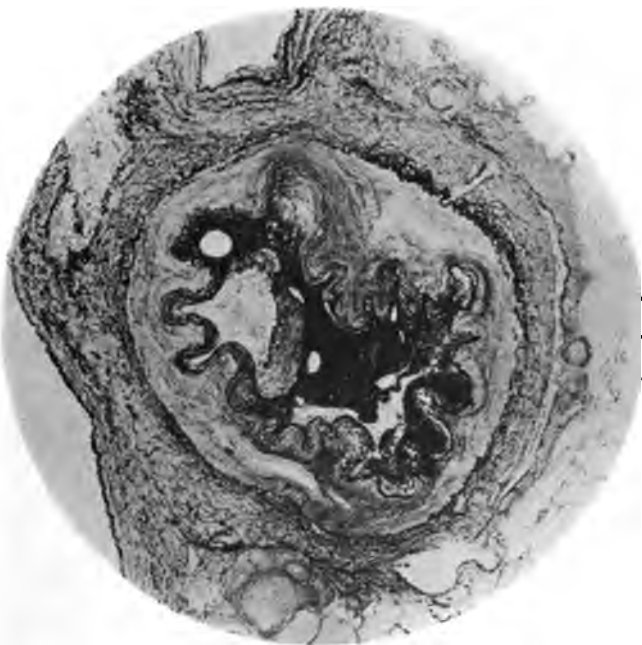


FIG. 1



FIG. 2

THE PATHOLOGY AND DIAGNOSIS OF SO-CALLED DIABETIC GANGRENE

By Leo Buerger

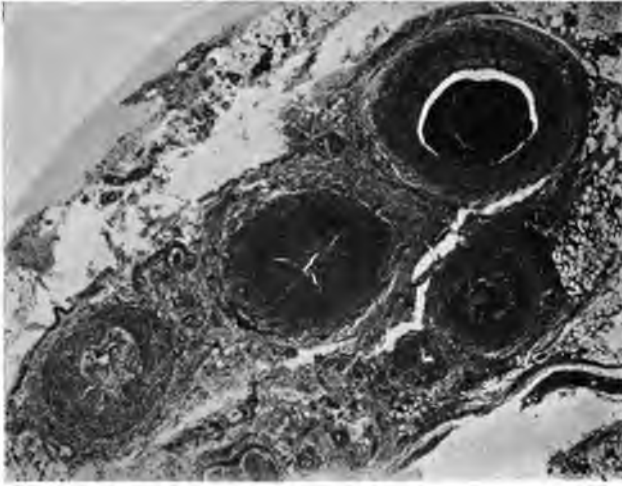


FIG. 4

THE PATHOLOGY AND DIAGNOSIS OF SO-CALLED DIABETIC GANGRENE

By Leo Buerger

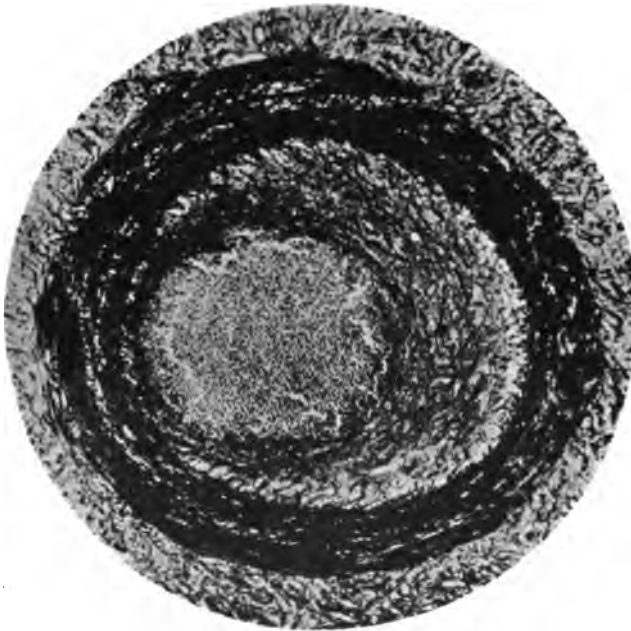


FIG. 8

Dorsalis pedis is open, but markedly atheromatous. The veins are all open; the external and internal saphenous are normal.

There is no periarteritis.

Summary:—A case of ulceration and beginning gangrene in a diabetic; extensive atherosclerosis with occlusion of many of the arteries, the veins being open. In short, this case is a typical example of the variety in which the atherosclerotic process is occlusive in nature.

As an example of somewhat less marked atherosclerosis, associated, however, with obliterative thrombosis, we have the following case:

R. A. "diabetic gangrene."

Right leg: ablation $6\frac{1}{2}$ inches above joint. The dorsum of big toe and adjacent surface of first metatarsal bone are the seat of beginning gangrene that has extended almost to the head of the metatarsal bone. This area is covered with a brownish green discolored skin beneath which lies a pool of fluid; when this necrotic material is wiped away, the tendon sheath of extensor muscles of toe and underlying bone are exposed. The necrotic material has insinuated itself beneath and around the extensor tendons of the second and third toes, downward as far as the base of these toes, and upward as far as the head of corresponding metatarsal bones. The epidermis covering the sole is easily stripped off over the anterior three quarters of the foot, exposing an irregularly quadrilateral gangrenous patch about the center of the foot. The appearance of this patch is like that described as occurring on the dorsum. It extends upward between the plantar fascia and muscle groups, apparently coming into contact with the first and second metatarsal bones and communicating with the gangrenous area on the dorsum.

Femoral artery is sclerotic and brittle. At one point near the lower end of femoral artery, there is a large ulceration of the intima about 3×7 mm. Just below this there is another ulceration with some undermining of the intima by extravasation of blood. The vessel itself is patent. The femoral vein is only slightly thickened and is patent.

Popliteal artery is patent throughout. Its wall is much thickened and somewhat calcareous, especially in the upper part, where there is an encircling band of calcareous material about 2 mm. in breadth.

The intima is smooth, but there is diffuse atheroma, this being especially marked about the orifices of smaller branches. The accompanying vein is only slightly thickened and is open throughout.

Posterior tibial, at its origin, and for a distance of about 11 cm. is patent; from this point on, there is an obliterating thrombosis of the vessel. The occluded portion is contracted. The vessel, as a whole, presents only a moderate degree of sclerosis, this being more marked from the point of thrombosis down.

Peroneal artery, at its origin, and for a distance of about 4 cm., presents a similar picture to the posterior tibial. In the rest of its course there is complete obliteration by an old, white, organized thrombus. The accompanying veins do not show as much thickening as posterior tibial, and are patent.

Internal saphenous vein shows marked thickening of wall, so that the vessel can be rolled beneath finger like a cord; it is, however, open throughout.

Anterior tibial shows a process similar to that involving the femoral though less extensive. Toward the lower end there is considerable narrowing of vessel as a whole, but the lumen is unimpaired.

Summary:—Gangrene associated with arteriosclerosis and atheroma of the femoral vessel; extensive obliterative thrombosis and atheroma of the posterior tibial and peroneal arteries with thickening of the internal saphenous vein.

Another instance of extensive arterial disease is seen in the following case:

A. B. "diabetic gangrene." Specimen—leg amputated at lower third of thigh.

Popliteal open; well developed calcific plaques.

Posterior tibial—atheromatous and calcified plaques which almost completely close the vessel.

External plantar—closed by atheromatous masses, also internal plantar.

Anterior tibial—pipe stem; partly open throughout; lower part of anterior tibial and dorsalis pedis practically closed by calcific atheroma.

Summary:—Evidently a case in which extensive calcification and

atheroma have almost completely closed the larger portion of the distal vessels.

ARTERIOSCLEROTIC GANGRENE

In this group we include a presenile and senile variety, the lesions being identical. In both there is well advanced atherosclerosis, which is in no way distinguishable from the vascular lesions associated with diabetic gangrene. In the presenile group that occurs in men of forty to fifty-five years of age, the calcific, degenerative process may be less advanced, but in the senile group of arteriosclerotic gangrene, we find exactly the same lesions that occur in the gangrene of diabetics. Characteristic of both the diabetic and arteriosclerotic groups is the involvement of the larger arteries, primarily by an affection that involves the wall of the vessel, secondarily by a lesion of occlusion, usually due to the production of degenerative atheromatous and calcific masses, more rarely by the presence of obturating, organized thrombi.

THROMBOTIC GANGRENE

There is another group of cases which occurs very frequently in patients after the age of fifty. Although the arteriosclerotic process is but moderately pronounced, an occlusive thrombosis suddenly develops, usually in the peripheral vessels such as the dorsalis pedis, plantars, anterior tibial and distal portion of the posterior tibial. In these cases we see mild or moderate lesions of atherosclerosis with a superimposed complete occlusion by what we term a "bland thrombus." Fig. 2 was taken from such a case in which amputation revealed only a very slight degree of atherosclerosis and a recent thrombosis of the distal vessels, the dorsalis pedis and posterior tibial.

THROMBO-ANGIITIS OBLITERANS

The name, thrombo-angiitis obliterans, was proposed in 1908¹ for that interesting group of cases of presenile gangrene previously described under the name, *endarteritis obliterans*. Pathological studies of some forty-five amputated lower extremities and of some twenty-five superficial veins affected by that stage of the disease known as "migrating phlebitis," have shown, that we are dealing here with

an acute inflammatory process involving the superficial veins, the deeper arteries or deep veins, followed by complete occlusion through the formation of red, obturating thrombi; and that a stage of healing follows through organization of the clot and resorption of the products of the acute inflammatory process.

The early or acute stage of the disease can be best studied in the superficial veins when these are the seat of the inflammatory thrombophlebitis. These lesions, as they are found in the exsected veins, are infiltration of the wall of the vessel with polynuclear leukocytes and occlusive thrombi (Fig. 3) with the formation of purulent or miliary foci of pus. Organization or healing then takes place, the purulent foci becoming changed into characteristic foci that closely resemble miliary tubercles. Later on, the exudative products in the vessel wall become absorbed, vascularization of the clot takes place, new vessels appear in the media, and the clot disappears, becoming canalized and vascularized. Finally, in the healed stage of the disease, the media of the vessel wall presents nothing remarkable, but the presence of new-formed vessels; the adventitia is thickened; a certain amount of periarteritis develops; the arteries and veins being firmly adherent to each other, and the lumen of the affected vessel is closed completely, the organized clot containing new-formed vessels or vascular spaces. These may give the occluded vessel the appearance that had been regarded for so many years as an obliterating endarteritic process.

When an artery and its *venae comites* are affected, various stages of the process may be found in the different vessels in the same sheath. Thus, in the posterior tibial depicted in Fig. 4, the artery is in the old or "healed" stage of the disease; so also is one of the veins. Another vein is in the acute and inflammatory stage, and still another vein is in an intermediate or healing stage where miliary foci are in evidence.

In short, we have here a specific entity in all probability of an inflammatory nature, leading to extensive occlusion of most of the arteries and often many of the veins of the lower extremities.

When the lesions of this disease are known, they cannot be mistaken either for endarteritis or for the atherosclerotic or arteriosclerotic processes that belong to the diabetic, senile and the pre-senile atherosclerotic cases.

EMBOLIC AND THROMBOTIC GANGRENE

This may occur either in cases in which the vessels are absolutely normal, or, somewhat more frequently, in cases in which an atherosclerotic process is present. As a complication and sequela of certain infectious diseases, embolic and thrombotic gangrene is not uncommon. We have seen a number of cases after severe pneumonia. One of the favorite sites for embolic or thrombotic processes is the popliteal artery, although an extensive thrombosis following embolism may occur in the femoral or even higher up. Both diabetic cases and cases not at all afflicted with this disease may be taken with sudden *occlusion* of arteries or veins.

In brief, therefore, an extensive atherosclerotic process is the usual pathological lesion not only in so-called diabetic gangrene, but also in the gangrene of senile cases, of the "arteriosclerotic" cases, of many cases of presenile arteriosclerosis, and in some of the cases of arteriosclerosis associated with thrombosis. In thrombo-angiitis obliterans, however, we have a distinct entity, not at all related to these diseases of the vessels previously described, in all probability of inflammatory nature, the inciting organism, if any be present, being unknown. As for the embolic gangrene, this may or may not be associated with disease of the vessel wall.

Regarding the pathology of the cases which we group as vasomotor or neurogenic, including Raynaud's disease, erythromelalgia and chronic acro-asphyxia, our own investigations have shown definitely that gangrene in such cases is not dependent upon organic vascular disease, both the arteries and veins remaining patent.²

The cases of diabetes in which trophic disorders or gangrene of the lower extremities develop, do not vary greatly from the senile or advanced arteriosclerotic cases, except that the complicating affection "diabetes" may augment the mortality, may influence the advent of coma, and may increase the tendency to phlegmon formation. Considerable diversity in the onset of the disease, manifold variations in the types of trophic disturbances and gangrene will be noted if a large series be collected for study. So too, in the occurrence of complications will variations occur.

The following is a history often obtained in these cases:

For months or years there has been some trouble in walking, such as pain in the calf, in the instep or in the ball of the foot on walking:

that is, intermittent claudication. Sooner or later, paresthesiae, burning sensation or pain will develop somewhere in the foot, usually at the site of impending gangrene. A dry patch of skin, an abrasion or a bleb will appear very frequently over the outer or inner border of the foot, near the head of the corresponding metatarsal bones, and will be transformed into a chronic ulcer or into a patch of dry gangrene. In other cases, a perforating ulcer will develop; and in still others, the process will take on a more fulminating type, gangrene of the moist type rapidly developing.

In any of these cases, when gangrene or trophic ulcers have made their appearance, secondary lymphangitis or phlegmon formation may rapidly take place.

A more protracted course is taken by those cases in which an ulcer of limited extent persists for weeks or months, may even heal, or give way to the formation of another ulcer in another portion of the foot, the latter lesion either terminating favorably, or more commonly leading to gangrene or infection.

The following extracts from the histories of some of my cases will illustrate some of the types encountered:

Rapidly developing wet gangrene.

I. Z., Aug. 8, 1914, female, age 54, was perfectly well until seven years ago when she complained of pruritus vaginae; sugar was found in the urine. Two months ago, after the removal of a callus on the sole of the left foot, a painful, indolent ulcer developed; this refused to heal. Recently there developed spontaneously a number of large blebs on the dorsum of the foot and over the toes.

Physical examination, Aug. 11, 1914. A perforating ulcer is present on the plantar aspect of the left foot. Over the dorsum of the same foot and just behind the middle toe, there are large blebs apparently filled with sanguinous fluid. The dorsum of the foot presents a peculiar bluish-red mottled discoloration, and when the blebs are opened, bloody fluid is obtained. The foot is markedly cold.

The right foot also shows a bleb over the tip of the big toe and extending over the plantar aspect.

Aug. 14. The ecchymotic area on the dorsum of the left foot is apparently extending; the second toe is cyanotic and cold.

Diagnosis: Impending wet gangrene.

In short, we have here a case, in which, superimposed upon a typically slowly developing, perforating ulcer, there supervened evidences of extensive wet gangrene. Interesting and characteristic for this type of gangrene are: Separation of the epidermis, the extravasation of bloody serum under the epidermis, cyanotic discoloration of the foot, coldness of the foot, coupled with the absence of pulsation in the larger vessels, including the dorsalis pedis, posterior tibial and popliteal.

More common are those cases in which the mortifying process is slow in developing, being attended sooner or later by an inflammatory or phlegmonous process.

H. G., male, age 62, Feb. 22, 1907. For about five years he has been drinking large quantities of water, and has to void frequently in large amounts. For three of four weeks he has had trouble with the big toe of the right foot, there being almost constant pain. The whole toe became black shortly after the development of a small, dried, blackish spot at the tip of the toe.

Physical examination: The right foot is somewhat swollen and reddened up to the ankle. The big toe shows the typical evidences of dry gangrene, extending up to the metatarso-phalangeal articulation, where there is an irregular line of demarcation. There are two small ulcerations of the dorsum of the toe which discharge a small amount of serous material. The pulses in the dorsalis pedis, posterior tibial and popliteal arteries are absent.

Pathological examination of the amputated limb shows gangrene of the big toe and purulent infiltration of the cellular tissues on the dorsum of the foot. Another phlegmon involves the plantar aspect in the region of the ball of the great toe. There is an extensive and intense arteriosclerosis.

In addition to the cases in which we either have rapidly developing gangrene or more slowly progressing dry gangrene with or without phlegmonous infiltration, there are other types in which merely a trophic ulcer is present or ulcers of small extent, that may or may not heal. A multitude of combinations can be expected, if we see a large amount of material. Characteristic, however, are these facts, that initiated by a period of intermittent claudication; or without any prodromal symptoms; or, following some trauma such as cutting a nail or a corn; or, after local infection; or, apparently without

cause, evidences of trophic disturbances make their appearance. These may manifest themselves in the form of a superficial ulcer, a perforating ulcer, or in the development of an area of dry or wet gangrene.

DIFFERENTIAL DIAGNOSIS

When we are confronted with cases of trophic disturbance or gangrene of the lower extremities, we will find that, for purposes of diagnosis as well as prognosis, a routine examination made according to a plan adopted by us some seven years ago will be of some value. Our scheme of physical diagnosis includes an examination of the following points: First, general appearance of the limb, including an investigation of the presence of trophic disorders or gangrene; second, appearance of the limb in the dependent position (presence of chronic erythromelia); third, presence or absence of ischemia or blanching in the elevated position; fourth, estimation of the angle of circulatory sufficiency; fifth, pulsation in the palpable vessels: iliac, femoral, popliteal, posterior tibial, anterior tibial and dorsalis pedis; sixth, the occurrence of an induced or reactionary rubor or erythromelia.

FIRST. The general appearance of the limb: In distinct contrast to the cases of thrombo-angiitis obliterans, the arteriosclerotic or elderly diabetic patient will show evidence of malnutrition of the limbs. The foot, as a rule, looks withered and shows evidence of some atrophy. The normal irregularities of contour produced by the extensor tendons and bony prominences, will be present, except if a complication such as infection or bilateral edema is present. In thrombo-angiitis obliterans there is a tendency to obliteration of these markings. There may be distinct pallor, even in the horizontal position. It is an evidence of poor circulation. Fissures, ulcers, perforating ulcers, gangrene, bullae, ecchymoses, impaired nail growth and gangrenous areas may be present.

Second, *on depressing the foot*, when there is no inflammation present, a red flush of the foot will be noted. This is a condition of *rubor* or *erythromelia*, as I have elsewhere termed it. It is brought about by a compensatory dilatation of the superficial capillaries, and is most characteristic of thrombo-angiitis obliterans. *It may be also*

present in the arteriosclerotic and diabetic cases. It seems to be an effort on the part of nature to make up for the impairment of circulation by virtue of dilatation of the superficial capillaries.

Third, *when the limbs are elevated*, blanching usually sets in rapidly, when mechanical interference with the circulation is at hand. *The extent of the blanching and the rapidity with which it appears are both valuable aids in the estimation of the amount of arterial disease.*

Fourth, *the angle of circulatory sufficiency**: In the diabetic or arteriosclerotic cases, the estimation of this angle is not of as great importance as in the cases of thrombo-angiitis obliterans. By elevation to the vertical we establish a blanched condition of the limb. If we then gradually depress the limb, about 30° at a time, we will note the *point at which the circulation will return*. If this be at the horizontal, we call "the angle of circulatory sufficiency" 90° . Normally, the angle should be 180° , that is, color should still be present when the leg is held perpendicular to the horizontal. The greater the impairment of the circulation, the more will we have to depress the limb before the evidence of arterial return will manifest itself in the integument. Thus, below the horizontal, the angle will be less than 90° .

In many cases of arterial disease, the estimation of this angle is a valuable adjuvant, not only in the recognition of the amount of occlusive disturbance, but also in prognosis.

Fifth, *absence of pulsation as an indication of arterial occlusion*: The femoral, popliteal, posterior tibial, dorsalis pedis and posterior tibial must be palpated in every instance. Absence of pulsation is, as a rule, an indication of occlusion at the point palpated.

Sixth, *reactionary hyperemia, reactionary erythromelia*: By this term, we mean an induced rubor that can be elicited in the foot when it is allowed to hang down, after a preliminary period of elevation to the vertical. It is a physiological phenomenon, that ischemia of a limb artificially produced by an Esmarch or Martin bandage, will be followed by sudden dilatation of the capillaries of the peripheral parts, when the circulation is allowed to return. We have made use of this well-known manifestation in the examination

*This term was proposed by the author some seven years ago in a Clinical Meeting of the Mt. Sinai Hospital Conference, but has not been published.

of cases, in which impaired circulation due to arterial occlusion is suspected. Particularly in cases of thrombo-angiitis obliterans, have we found it applicable, but also in other cases of organic vascular disease. Thus, in early cases of arteriosclerosis, as well as in thrombo-angiitis obliterans, we may find that, after preliminary elevation, and consequent depression of the limb to the dependent position, a very striking and intense rubor appears. This is practically diagnostic of arterial occlusion, because it seems to indicate that blanching has been produced. In early cases, it is especially valuable, for it may be present long before the chronic condition of "erythromelia" or rubor in the dependent position develops.

In short, for all cases of suspected arterial occlusion, a routine examination, which has for its purpose the production of objective evidences of the effects of arterial occlusion, and which includes the palpation of the arteries themselves, is of extreme importance in diagnosis.

Having diagnosticated, then, the presence of mechanical interference with the circulation, by the demonstration of the signs and symptoms discussed, we must make a differential diagnosis in all cases between the neurogenic vasomotor and trophic diseases that may be attended with gangrene, and those affections that depend upon organic arterial disease. In a discussion of the subject of diabetic gangrene, the vasomotor cases need but slight mention, for the differential diagnosis is not difficult. For our purposes it is more important to distinguish that large group of cases which we have termed *thrombo-angiitis obliterans*, from the arteriosclerotic gangrene, for, diabetic gangrene is but a subdivision of the atherosclerotic or arteriosclerotic type.

If we pass in review the characteristic features of thrombo-angiitis obliterans, which I have discussed in detail elsewhere,³ we could summarize these as follows: The disease occurs, as a rule, in young men between the ages of twenty and thirty, although the symptoms may not appear until as late as forty or fifty, or even at a later age. More than 99 per cent. of the cases seem to occur in Polish, Galician, Russian and Austrian Jews, females being practically never afflicted. One of the first symptoms is pain in the foot or in the ankle, more commonly in the calf on walking, or a feeling of tiredness that leads the patient to take frequent rests, and often

suggests to him to seek the advice of an orthopedist. After a prodromal period of weeks or months and sometimes years, in which the symptom of intermittent claudication may be the most prominent, there develops either spontaneously, or after the cutting of a nail, callous or after injury, some evidence of trophic disorder. At the tip of one of the toes, or at the outer border of the foot under a nail, or in the web between the toes, a dry patch of skin which soon blackens into an area of gangrene may develop, or a hemorrhagic bleb appears and is followed by the development of an ulcer; or a painful fissure is produced. This is the second stage of the disease, which may be regarded as that of trophic disorders and impending gangrene. About this time, or even sooner, a peculiar reddish blush of the foot takes place which is intense in the dependent position, and disappears upon elevation of the limb. We have termed this condition of rubor *erythromelia*. Because of its constancy and intensity it is characteristic for this disease, although also associated with arterial occlusion due to other causes, such as atherosclerosis. About this time, there develops intense pain, either in the region of the ulcer or gangrenous patch or throughout the leg. The pain may become so intense at night, that sleep is impossible, the patients being robbed of sleep for days and weeks at a time. From this point on, the disease may take a varied course. Trophic disorders may last for weeks, months, or even years without making much progress, may heal spontaneously, or give way to dry gangrene, amputation being the issue. Sooner or later, however, in most of the cases, gangrene of one limb becomes either so extensive or the pain becomes so excruciating, that the extremity cannot be saved.

In short, after a prodromal period of indefinite pain or intermittent claudication, there develop symptoms of trophic disorder and manifestations referable to impaired circulation, leading finally to gangrene and amputation.

Another characteristic feature of the disease (to which I called attention in 1909⁴) is the occurrence of a characteristic migrating phlebitis involving the superficial veins of either the lower or upper extremities, preferably seeking the territory of the internal and external saphenous. Extending over a period of weeks, months, or even years, we see the appearance of painful nodosities and of elongated, inflamed cords in the skin, manifestations which indicate

the involvement of superficial venules or larger veins with the disease, thrombo-angiitis obliterans. The exsection of such veins followed by careful histological examination has revealed to us that *there is a specific morphological picture characteristic for thrombo-angiitis obliterans, which can be demonstrated not only in the superficial veins, but also in the deep vessels of the amputated limbs.* This specific picture, not discoverable in any other thrombotic disease, is preceded by an acute inflammatory stage (Fig. 3) which would lead us to assume that we are dealing here with a disease of infectious origin.

Although the recognition of the disease, thrombo-angiitis obliterans, will rarely be difficult in the young, the older patients may present difficulties in differentiation from arteriosclerotic disease. The diagnosis of thrombo-angiitis obliterans must depend upon (1) the racial (Hebrew) and sex (male) predilection; (2) the early involvement of the lower extremities; (3) the early symptoms of pain or intermittent claudication; (4) the presence of migrating phlebitis; (5) evidences of pulseless vessels; (6) the presence of blanching of the extremity in the elevated position; (7) the existence of rubor in the dependent position; (8) the relation of the hyperemic phenomena to posture; (9) the absence of simultaneous, symmetrical involvement; and (10), the slow, progressive chronic course terminating in gangrene.

In the arteriosclerotic (diabetic) gangrene the general appearance of the limb will be different. The foot will have a somewhat atrophic appearance, except in the stage of infection. The gangrenous process advances more rapidly, is more frequently of the moist type, more often associated with phlegmon formation and extensive sloughing, more frequently preceded by a perforating ulcer. The erythromelia, if present, is less marked, less distinct; there is no migrating phlebitis, and there are distinct evidences of arteriosclerosis, often recognizable in the condition of the dorsalis pedis or other vessels. Purely vasomotor symptoms are rarely present. When the arteriosclerotic gangrene occurs in patients between forty-five and fifty-five years of age, a differential diagnosis from thrombo-angiitis becomes difficult and often impossible. For, in certain instances, thrombo-angiitis obliterans may have existed without symptoms since early adult age, and may have become spontaneously cured, the

clinical effects of the obturation of the arteries having been completely compensated by the establishment of adequate collaterals. In such cases, gangrene may occur later in life, not because of the former thrombo-angiitis obliterans, but because of the subsequently engrafted arteriosclerotic disease. Pathological studies of amputated limbs have definitely shown that such a combination can occur.

A differential diagnosis from the true vasomotor conditions will be rarely an arduous task. It will suffice to take Raynaud's disease as an example and point out its chief characteristics.

In Raynaud's disease we will note the following features: A sudden onset of the first stage of local syncope or regionary ischemia involving usually the fingers, more rarely the toes, and occasionally the margins of the ears or the tip of the nose with coldness and blanching; associated sensory phenomena, paresthesia, and pain; a comparatively short duration of the vasomotor and sensory manifestations, their intermittent character with return to normal between the attacks; the symptoms of local asphyxia attended with local depression of temperature and swelling of the parts involved; the disappearance of the asphyxia with substitution of reactive hyperemia and a third stage of dry gangrene. Characteristic for this disease as well as for the cases of scleroderma and sclerodactyly is the striking atrophy of the ends of the distal phalanges. The changes in the bones can be well demonstrated by Röntgen-ray examination, atrophy and disappearance of large portions of end-phalanges being distinctive and diagnostic features. In our own experience the alterations in the bones could be detected early in the disease, probably developing simultaneously with the other trophic disturbances.

CONCLUSION

For purposes of clinical diagnosis, prognosis and for a correct understanding of the pathology of the various conditions complicated with gangrene of the lower extremities, it is expedient to classify the cases into two large groups: those in which the trophic lesions depend upon organic disease of arteries or veins, and those in which the nutrient vessels are intact. Pathological studies have led us to the opinion that one large group of cases—designated as thrombo-angiitis obliterans—must be separated from a second large group—athero- or arteriosclerotic disease,—because the pathological

lesions are distinctive in each group. In the latter group belong the cases of so-called diabetic gangrene, as well as the "senile" and some of the presenile cases. From the clinical standpoint, a classification in which vasomotor cases, including Raynaud's disease, erythromelalgia, etc., are recognized as being independent of organic arterial disease, and in which thrombo-angiitis and arteriosclerotic gangrene are conceded to be distinct entities associated with characteristic and extensive vascular disease, will give the best opportunity for correct diagnosis.

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THE DIAGNOSIS OF ABNORMALITIES OF MYOCARDIAL FUNCTION

By T. STUART HART

Assistant Professor of Clinical Medicine, College of Physicians and Surgeons,
Columbia University; Visiting Physician, Presbyterian Hospital
New York

IV. TACHYCARDIA—THE ACCELERATED HEART.

A heart rate of abnormal rapidity is one of the most frequent phenomenon observed by the physician. For purposes of the present discussion one may classify all such cases in two groups:

I. ACCELERATED HEARTS.

II. PAROXYSMAL TACHYCARDIA.

The main clinical feature which distinguishes these groups is the manner in which the transition from the normal to the abnormal

rate is accomplished. In the case of the *accelerated heart* the transition from the slow to the rapid and from the rapid to the slow rate is gradual; in a very brief period the heart cycle may become so shortened that the rate per minute is increased 50 per cent., and yet, as observed by palpation or auscultation, the length of any two successive cycles is so nearly identical that neither the finger nor the ear is able to detect the minute differences which go to make up the change.

In the *paroxysmal tachycardia* the onset and the offset of the change in rate is abrupt and the observer and even the patient is usually able to detect the sudden transition without difficulty.

THE ACCELERATED HEART

ETIOLOGY AND PATHOLOGY

It has already been pointed out that the rate of the normal heart is not fixed, but varies with the needs of the body at any particular moment. This rate adjustment is brought about through the regulatory mechanism of the extra cardiac nerves. In the conditions now to be considered the underlying factors are many and complicated, but we may recognize three important elements which individually or in association may produce an abnormal acceleration of the heart:

(A) *The outside demands on the heart may be excessive.*

A full discussion of the demands on the heart which originate outside of the cardio-regulatory nervous mechanism and the cardiac tissues themselves, the nature of such demands and their modus operandi, important and interesting as they are, would lead us outside of the limits which we have set in these papers devoted to the subject of myocardial function. However, this outside call for increased cardiac activity must never be lost sight of in analyzing the response of the cardiac tissues to these demands. A simple illustration of the response of the heart to increased demand is seen in the effect of work. As a general rule it may be stated that the response to physical exertion of an individual with a good myocardium is shown in an increased blood pressure. One with a defective myocardium shows an abnormal acceleration of the heart rate. With a normal heart muscle under efficient regulation and a normal vasomotor tone, moderate exercise causes an increase of cardiac rate,

but with rest the rate should return to its usual level in the space of a very few minutes. That the demands of exercise produce an intrinsic physiological effect on the myocardium is evidenced by the fact that the normal electrocardiogram constantly shows under such stress definite though small changes; in addition to the shortening of the diastolic period (*T-P*) there is an increase in the size of waves *P* and *T* and a deepening of *S*.

(B) *The extracardial nerves may be at fault in their regulatory capacity.*

It is quite evident in certain accelerated hearts that the fine nervous adjustments are unbalanced. The activity of the vagi are depressed or there is an excessive activity of the accelerators, such a lack of balance mainly affects the heart through its pacemaker, the sinus node. This is probably the mechanism of the rapid changes of rate in emotional conditions, the so-called "labile pulse" of neurasthenics and the more persistent rate increase in certain organic lesions of the central nervous system and of the peripheral nerves supplying the heart.

(C) *The heart muscle may be defective* and responds to normal outside demands with abnormal acceleration. The direct application of heat to the myocardium is known to increase the cardiac activity. Bacterial and chemical toxins set free in many of the infectious diseases are recognized as efficient agents in causing functional or organic changes of the myocardium, which are the basis of a response in rate out of proportion to the stress.

While we can sometimes designate one of these particular factors, excessive outside demands, defective nerve regulation or myocardial damage, as the cause of the increased heart rate, the problem is usually more complicated. No doubt frequently two or all of these elements play a part. In the present state of our knowledge we are often at a loss in deciding which link in the chain is at fault, and, if more than one, their relative importance.

Fever is nearly always accompanied by an acceleration of heart rate, and so uniform is this phenomenon that the well known Liebermeister's rule of an increase of 8 pulse beats for each degree of temperature above the normal is found approximately accurate, albeit, with many exceptions. Whether this is brought about by the increased temperature of the blood passing through the heart, or by the

chemical action of associated toxins on the regulatory nervous mechanism, or on the cells of the cardiac muscle, is undecided.

The increased heart rate of *shock* is undoubtedly due to local or general vaso-motor disturbance with its reflex demands on the heart to maintain an adequate blood pressure. A similar explanation seems probable for *Graves' disease*, and the excessive administration of thyroid extract in which the evidence points to the damaging effect of toxins on the vaso-motor apparatus, rather than the heart muscle. The "labile pulse," wide pulse pressure, flushing, local sweating and tremors characteristic of this disease suggest that the toxins chiefly attack the sympathetic nervous system, possibly incidentally producing a hypertonus of the accelerator nerves, and probably act on the heart muscle only in an indirect manner. *Pregnancy* probably has only a reflex effect on cardiac activity.

Exhausting diseases (tuberculosis, etc.) and *convalescence from wasting diseases* (typhoid, etc.), nearly always show some degree of increase pulse rate. Each one of these conditions, febrile or afebrile, with toxic and nutritional disturbances may affect the outside demands on the heart, the functional balance of the extracardial nerves, or the cardiac muscle, and in each instance the effort should be made to determine and apportion the relative responsibility of each of these factors in the acceleration of the heart. The *severe anemias*, high grades of chlorosis, marked secondary anemias (as in malignant disease), and the primary pernicious forms are invariably associated with an increase in heart rate. In the extreme grades of anemia the cardiac muscle shows an advanced degree of degeneration with fatty infiltration and hemorrhages,¹ so that we have little hesitancy in ascribing the altered heart activity to the direct toxic or nutritional effect on the myocardium.

In *valvular disease* the mechanical defect must be considered. The volume output is unusual and the normal bodily calls for blood are met by an increased heart rate. In the majority of these cases, however, the disease which was the agent in distorting the valves has also injured the myocardium and this, in association with the change in cardiac tone resulting from dilatation and hypertrophy, are important influences in modifying heart rate. Changes in the myocardium are produced by *acute rheumatic fever* and *other infectious diseases*

1. Lazarus.—"Pernicious Anemia," Nothnagel's Practice, Phila., 1906, p. 283.

with a resulting acceleration of heart rate. These changes may be chemical with no demonstrable histological abnormality, or there may be fatty degeneration and fibrous replacement, so that we meet with many degrees of functional impairment.

MECHANISM

The main link in the mechanism through which the increased rate of the "accelerated heart" is produced is the "sinus node," the normal pacemaker of the heart. Here the fundamental properties of "stimulus formation" or "excitation" or both, become heightened. This change may be intrinsic, that is to say, the chemical processes of the muscle cells of the node are so changed that they form and explode stimulus material more rapidly, or the change may be brought about by the modifying impulses showered on the node by the extracardial nerves. The sinus node is particularly influenced by impulses brought to it by the right vagus and the right accelerator.²

The distinguishing feature of the "accelerated heart" is that the sinus node retains its function as the pacemaker of the heart. This is shown by the graphic records which indicate that the impulse formation arises at the normal point and spreads through the auricle, the bundle of His and the ventricle in a normal orderly fashion. There are several facts, however, which indicate that, in these "accelerated hearts" other portions of the musculature may have their properties of "stimulus formation," "excitability," and perhaps also "conduction" heightened. It is known that the fibers of the left vagus and of the left sympathetic are in the main distributed to portions of the heart below the sinus node,³ and experimental evidence indicates that cutting the left vagus and stimulating the left sympathetic have a considerable effect in increasing the heart rate. Again in certain "accelerated hearts" it may be seen that systole, which in the normal heart has a very constant length, is shortened. This is only conceivable on the ground that one or more of the fundamental properties of cardiac muscle mentioned above are quantitatively changed.

The principal change from the normal in the cardiac cycle of the accelerated heart is a shortening of the diastolic period. From this it follows that the rest period of the heart is curtailed and the time

2. Robinson and Draper.—*Jour. Exp. Med.*, 1911, XIV, p. 227.

3. Cohn and Lewis.—*Jour. Exp. Med.*, 1913, XVIII, p. 739.

allowed for the recovery of the property of "contractility" is considerably less than in the heart working at the normal rate, hence the contractile power is less. Furthermore there is less opportunity for the heart to receive its normal quota of blood, hence the volume output is smaller. It follows as a result of these two factors that the pulse is smaller in volume and of diminished force.

IDENTIFICATION

Little need be said of the clinical recognition of the "accelerated heart;" the pulse may be counted either by palpation at the wrist or perhaps more accurately by auscultation at the apex. If one is pres-

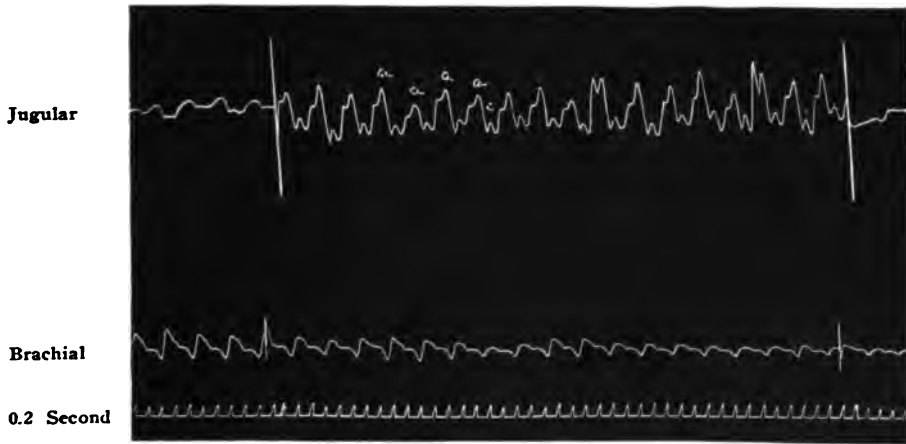


FIG. 1

Accelerated heart. Rate 145. Patient suffering from rheumatic myocarditis.

ent during the change from a slow to a faster rate this is best detected by counting the pulse in 10 second intervals, omitting every other 10 seconds. Neither the finger nor the ear can detect the small differences in the lengths of the successive diastolic periods, but the variations in length of the cycles separated by considerable periods is easily made out. The volume output of the heart is usually somewhat diminished with the acceleration of the rate and the consequent diminution in the peripheral arterial wave may be quite evident.

The *polygram* of the accelerated heart conforms to the normal

except that the diastolic period is shortened. This is at times so marked that the *a* wave may be superimposed on the preceding *v* wave. The jugular tracings (Figures 1 and 2) show a normal sequence of waves, *a*, *c*, *v*. Figure 1 is a record of a girl, 15 years of age, suffering from rheumatic myocarditis and adherent pericardium. The rate at the time the record was taken was 145 and the rapidity was in part due to excitement, as her pulse at rest was commonly 120. The slightest physical exertion at this time would send her pulse to 160, suggesting a marked instability of the sinus node.

In Figure 2 is shown a tracing of a case of Graves' disease; the rate is 138. It is evident from the jugular tracing that the normal

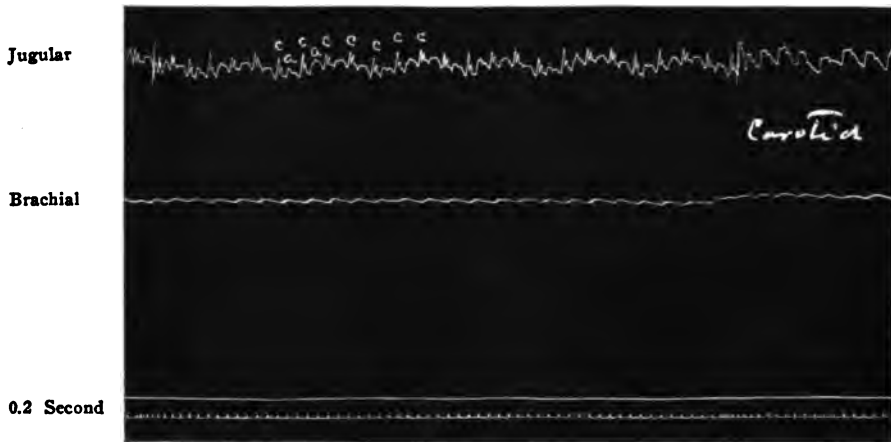


FIG. 2
Accelerated heart. Rate 138. Case of Graves' disease.

pacemaker is in control and that the rapid rate depends upon the shortening of the diastolic period.

Electrocardiograms of accelerated hearts are presented in Figures 3 and 4. Figure 3, from a case of Graves' disease, shows a short diastolic period, but the sequence of waves is normal. Figure 4 was obtained from a case of cerebral hemorrhage, a few hours before death. The diagnosis was confirmed by autopsy and it seems clearly a case in which the nervous regulatory mechanism is at fault. The *P* and *T* waves in this record overlap. Careful measurement sug-

gests that the earlier of the two peaks represents the auricular contraction which occurs before the preceding ventricular systole is completed. This curve simulates quite closely the records obtained ex-

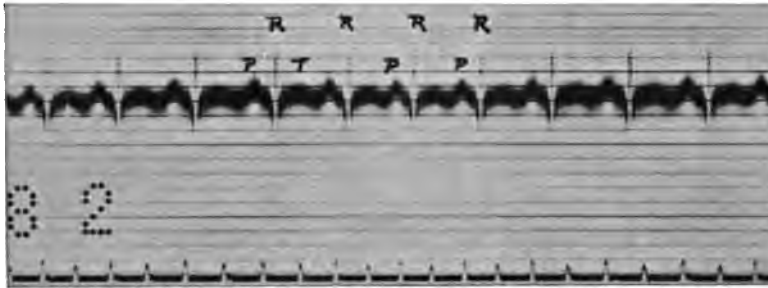


FIG. 3

Accelerated heart. Rate 148. Case of Graves' disease.

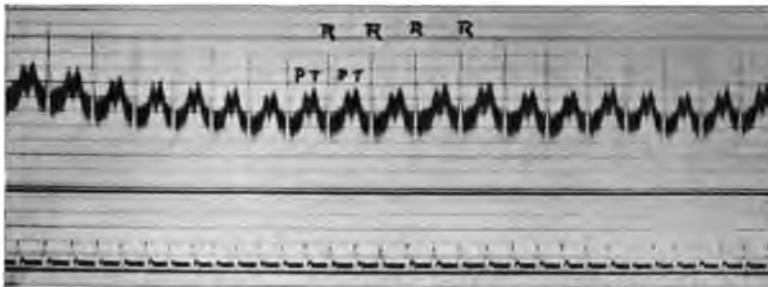


FIG. 4

Accelerated heart. Rate 184. Terminal tachycardia of cerebral hemorrhage.

perimentally during the stimulation of the right sympathetic ganglion by Rothberger and Winterberg.⁴

THE CLINICAL SIGNIFICANCE AND PROGNOSIS

of the accelerated heart depend on the underlying condition and to determine this, the responsibility of excessive outside demands, lack of balance between the elements of the nerve regulatory mechanism

4. Archiv f. d. ges. Physiol., 1910, CXXXV, p. 557, Fig. 18, d.

and defects of the myocardium, must be correctly apportioned. In general one may say that excessive outside demands and unbalanced nerve control acting on a heart with its myocardium intact, are usually more readily corrected, and hence of less serious import to the patient than when the heart acceleration depends upon an intrinsic defect of the myocardium. But even a normal myocardium may be worn out by the excessive activity induced by extracardial conditions, and a defective myocardium properly handled may recover full functional efficiency. The tests by which we may gage the integrity of the heart muscle and its reserve force will be discussed in a later paper.

A SIMPLE SUBSTITUTE FOR THE WASSERMANN REACTION

By G. ARBOUR STEPHENS

Hon. Physician, Royal Cambrian Institute for the Deaf; Fellow of the Medical Society of London; Late Lecturer on Biology,
Swansea Technical College

Swansea, England

Under the heading of "Distilled Water Versus Salvarsan in the Treatment of Syphilis," in the British Medical Journal for April 5, 1913, I tried to show that excellent results can be obtained in the treatment of syphilis by the subcutaneous injection of distilled water, and as it is unattended by any danger, therefore, on account of its ease of application, such a treatment ought to appeal to the general practitioner.

Since I published that article I have had a large number of other cases which have responded with the same readiness and with the same result. Distilled water, like salvarsan, must be used in conjunction with the internal administration of mercury, which I always administer in the form of colloidal mercury, and especially that made by Merck of Darmstadt.

In the Practitioner for September, 1910, and The Dublin Journal of Medical Science for June, 1911, I gave the results of some of my work on colloids and surface tension, and as the result

of that work I determined to try the effects of distilled water in syphilis, and, fortunately, with good results.

Following up such a successful issue of a treatment arrived at on theoretical considerations, I decided to compare the response from a surface tension point of view of healthy and syphilitic blood to various solutions.

Amongst these solutions was ammonium chlorid, which has the unusual effect of raising the surface tension. In all these experiments it is very important to have all the reagents pure and the glasses clean, otherwise any impurity will negative the rise that ought to take place.

The best solution of ammonium chlorid to use is one of about 12 per cent., and the way to carry out the test is as follows:

Clean the lobule of the ear with petrol, prick it and draw off a sufficiency of blood in a hemocytometer pipette, that is, up to the half mark, and fill up to the remainder of the pipette with a 12 per cent. solution of pure ammonium chlorid in distilled water.

The blood and solution should be thoroughly mixed, and at once examined under the microscope. Any delay is dangerous, for evaporation takes place, and the ammonium chlorid crystallizes out very readily and spoils the test.

Examined in this way, the red corpuscles show a marked change, for in healthy blood the darker center tends to contract or crinkle up into an irregular mass, whereas in syphilitic blood the darker center tends to expand in some cases out toward the circumference of the corpuscle.

The advantage of this test is its simplicity, enabling it to be carried out by any medical man, and it only takes three minutes to perform, whereas the Wassermann is complicated and expensive and requires a practitioner specialized in the work.

There are, of course, gradations in the results from complete contraction to the point when doubt arises, a point that is frequently arrived at even with the Wassermann.

In this connection I would mention an interesting case which I saw fourteen days ago for the first time.

The man, aged 23 years, had just got over an attack of gonorrhea, but felt unable to follow his occupation on account of disinclination for work and depression. I examined his blood, which

seemed to me to be satisfactory, and I gave him a good nerve tonic, but without any benefit. I again tested his blood with the ammonium chlorid, but this time the result was marked and I forthwith injected 10 c.c. of distilled water under his skin, and ordered him 30 minims of colloidal mercury. He returned in three days for a second injection, when his appearance had altogether changed, and his depression had disappeared. So much did he feel better that he was arranging to go back to London on the following day.

I usually inject 10 c.c. of distilled water twice a week for six weeks and then give a rest.

FACTS AND FALLACIES CONNECTED WITH THE CLINICAL PATHOLOGY OF THE ACETONE BODIES

By HEINRICH STERN

New York

While in many instances of acidosis B-oxybutyric acid is encountered in undue amounts, this is by no means the only low fatty acid that contributes toward the acid intoxication. The other members of this series, proprionic, valeric, capric, enanthylic, caprylic, pelargonic and capric acids are probably as important in the production of acidosis as are the members of the butyric acid group themselves. Furthermore, besides acetone (C_3H_6O), the ketone yielded by acetic or aceto-acetic acid, the ketones formed from the successive members of the fatty acid series, differing from one another by twice CH_2 , undoubtedly participate in the production, or are concomitants of the clinical pictures that are erroneously ascribed to the acetone bodies or their direct progenitors. Such ketones are propione ($C_3H_{10}O$) yielded by proprionic acid, butyrone ($C_7H_{14}O$) from butyric acid, and valerone ($C_9H_{18}O$), a product of valeric acid. The close chemical relationship of the successive members of the fatty acid series and that of their respective ketones, and the facts that they are, to the greater part, volatile liquids which are readily intermiscible and are subject to the same chemical reactions, give strength to the assumption that one member of the series of fatty acids or ketones may preponderate in a given case, but that it is hardly probable that these single members are present to the exclusion of all the

others. Originating in the organism from practically the same source or sources and being affected by the identical chemical influences, it is obvious why more than one of the lower fatty acids and more than one of their ketones are apt to occur at a time, and why the phenomena of acidosis, which are by no means invariable and uniform, must of necessity be the result of the conjoint activity, or be the concomitants of various fatty acids and various ketones.

It is probably true that there is no case of general acidosis in which acetone or its immediate forerunner, diacetic acid, cannot be demonstrated in the urine; this, however, is no conclusive proof that other ketones or their corresponding fatty acids are not found associated with the former. It is simply the readiness with which acetone and diacetic acid are detected in the urine that has given them a clinical prominence which, in reality, they do not deserve. Were the other ketones and fatty acids as easy of demonstration, the acetone bodies and their direct progenitors would not be exclusively held responsible as the *materia peccans* or be considered the main pathological product in acidosis.

While the occurrence of acetonuria may, therefore, furnish convincing evidence of an imminent or already established acidosis, it is in itself no proof that other members of the low fatty acid series have not participated in establishing this abnormal state.

Although acetone and other ketones appear very promptly in the urine, the amount in which they occur therein is no direct or un-failing indication of the intensity of the degree of acidosis. This is particularly the case in infants and young children, in whom the very volatile ketones leave the body principally through the medium of the expired air. As a rule, long before the ketones can be demonstrated in the urinary secretion, they have manifested their presence in the air exhaled by the little patient. Of course, the same series of ketones is also contained in the air from the lungs expelled by adult patients with acidosis, but here these substances occur in infinitely smaller amounts, their greater part being excreted by the kidneys. A certain quantity of these volatile bodies, especially in the very young, is also apt to escape through the mouth as one of the terminals of the digestive tract. In this case, it is very likely that they have derived from the alimentary canal where they were produced. The best, and probably the only positive, manner

to determine the presence of ketones in the expired air is by means of well-trained olfactories. This olfactory testing, at any rate, is the only clinical method that is at our disposal. The ketones cannot alone be recognized by the use of olfaction, but their amounts be roughly estimated by constructing an olfactometer for a given case.*

We find it in the text-books generally that the breath in acidosis is sweetish, fruit-like. As far as this pertains to the adult, this is certainly true in a certain proportion of the cases. In children, on the other hand, in whom the far greater part of the pathologically increased volatile acids and ketones leave the body by the expired air, this has always a distinct, and occasionally a penetrating, odor of impure rancidity. This odor, to be sure, is far from being sweetish, and is certainly not resembling that emanating from fruit or chloroform. A whiff of it, once conveyed to the olfactory center, will always linger in the memory. This odor may be likened to that of a mixture composed of about sixty per cent. of butyric acid, thirty per cent. of valeric acid and ten per cent. of acetic acid. The proportions, however, are not definite, and the odor varies within certain limits. In some cases, particularly when diabetes is present, the odor is not seldom extraordinarily vile. I must confess that the ketone-laden breath almost overpowered me in a few instances, and in the case of a four-year-old diabetic girl the odor was so strong and lasting that it still could be noted in my office hours after she had left. Blessed are those who, being compelled to be about such little patients, are not endowed with a keen sense and appreciation of smell.

This rancid odor is by no means to be ascribed to that caused by the souring of milk in the stomach. In the first instance, the odor is solely communicated by the expired air in the preponderating majority of the cases; secondly, it has very little in common with the odor of curdled milk; thirdly, it endures and cannot be

*It is a pity that so many undertake the study of medicine without attempting to uniformly train and develop all their five senses. The most neglected of the senses, as far as their employment in clinical medicine is concerned, is that of olfaction. The average individual, from whom the physician makes no exception, relegates the important function of smelling to the dog, deeming himself superior to that what he is wont to call "a dog's trait." The sense of smell, however, will detect the presence of infinitesimal amounts of substances which are not at all shown by either the microscope or the test tube reaction.

modified by medicaments introduced by mouth or rectum, and, fourthly, it is present when the stomach is entirely empty, when the little patient had not ingested milk for days and, also, when there is total abstinence from food.

It seems that only when the pathologic excess of the volatile acids and the ketones is very great a certain proportion of them find their way into the urine. When, therefore, the urine of infants and young children exhibits acetone or its progenitors, the case cannot *a priori* be counted among the milder ones. Under such circumstances, one always has to deal with a certain degree of intoxication. In adult individuals where most of these pathologic amounts of fatty acids and their intermediary substances are eliminated from the economy through the medium of the urine, a ketonuria is invariably of a far less pathologic significance as one of similar intensity-degree in infants. Thus, it may happen that the milder degrees of acid intoxication in children, those cases not manifesting themselves by pathologic amounts of the acetone bodies in the urine, are either entirely overlooked or regarded as something else.

The ketones may also be removed by way of the bowels. In this case they may be excreted with the feces-or they may be expelled with the intestinal gases. In all cases in which acetone was found in the feces themselves, the urine also contained large amounts of this substance. The occurrence of acetone in the feces seems to point to its local, its enterogenous, production.*

It has already been stated that the ketones, on account of their great volatility, are readily absorbed. If their absorption through the lacteals does not ensue, either a catarrhal affection accompanied by diarrhea is interfering with it, or the acetone substances are not yielded until the residual ingesta containing the lower fatty acids have reached the large bowel. It is my clinical conviction that a structurally or functionally diseased cecum, hindering the

*Acetone in the stools may be detected in the following manner: The fresh feces are first well diluted with water, acidified with acetic acid, and then distilled. The distillate (10 c.c.) is treated with a solution of iodine in ammonium iodide; this results in the formation of iodoform and a black precipitate of nitrogen iodide. The latter gradually disappears on standing, thus rendering visible the iodoform. This test is reliable, as it excludes disturbing factors and sources of error like alcohol and aldehyde.

free absorption of water, is the frequent seat of the formation and retention of enterogenous ketones.

The observation that the ketones may be expelled with the intestinal gases was made by me. Their presence can clinically only be demonstrated by the sense of smell. A decided ketone odor in the intestinal gases I have noted in three diabetic children, aged five, nine and ten years, respectively. The expired air and the urine of these patients contained large amounts of acetone and its associated substances.

In other cases, the intestinal gases also contain ketones. However, their presence is not as readily discernible, as they are admixed with the other gases of fermentation and putrefaction. One cannot err often by maintaining that the butyric-valeric odor of the flatus in the presence of ketone expiration and ketonuria is occasioned by the occurrence of the same ketones in the intestines.

While it is likely that the ketones expelled with the flatus are of enterogenous production, one cannot entirely dismiss the thought that they may possibly be the consequence of metabolic disturbances beyond the intestinal wall. The large quantities of acetone bodies in the expired air and the urine of many patients would point to this. At the same time, it must not be lost sight of the possibility that many cases of enterogenous acetone production do not concur with an acetonuria, that the ketones may leave the body at the termini of the alimentary canal, and that the entire pathologic process may be confined within the latter. The badly smelling flatus and stools in such cases may, and often are, caused by the low fatty acids and their products of decomposition.

KETONES OF ENTEROGENOUS FORMATION

By HEINRICH STERN

New York

For clinical purposes I have subdivided the cases of acid intoxication into *accidental* and *catabolic* types. The former comprise all the instances in which the ketonuria is of supposedly intestinal origin, the latter those in which the ketones are assumed to be the direct or mediate result of incomplete or perverse processes beyond the stage of anabolism.

Since the publication of my book on the autotoxicoes,* in which the pertaining theories and data were discussed at greater length, very little of import has been added to our knowledge concerning the ketones and acidosis. This is especially true as regards the accidental or intestinal acetonuria. As to the clinical side of this question, it seems almost that the pediatricists are considering it their exclusive domain. They report case after case, accusing a supposed acidosis as the causative factor of the trouble, but forget or are unable to furnish positive proof thereof.

Moreover, the clinicians are not at all certain whether an acute acid intoxication, to which the various non-diabetic symptom-complexes in children are ascribed, is of enterogenous or catabolic production. Some even go so far as to deny the possibility of the intestinal origin and the localized activity of the *materia peccans*, but apply just the same remedies, they claim with success, which do not exert any influence at all beyond the intestinal wall.

Of course, it is clinically not always discernible where the intestinal production of the ketones ends and their catabolic formation begins. Moreover, it is quite feasible that certain instances of ketonemia may be due to an association of intestinally and catabolically developed substances.

A differential diagnosis may sometimes be entertained on the basis of the following points:

	<i>Intestinal Acidosis.</i>	<i>Catabolic Acidosis.</i>
Age.	Mostly in infants and young children.	Most frequent after middle life.
Mode of onset.	Frequently by some form of gastrointestinal derangement.	Gradual.
Duration.	More or less transient, but tending to recurrence. Ceasing with the underlying disorder.	Mostly lasting like its substrate.

*The Autotoxicoes: Their Theory, Pathology and Treatment. Chicago, 1906.

Intestinal Acidosis. Catabolic Acidosis.

Significance. Perverse disintegration and Alkali deficit in tissues; excretion of fatty sub- (anomalous) disintegration of body fat. substances in alimentary tract.

Body weight. No, or no marked, influence. Gradual loss.

Alkali therapy. Not producing any improvement. Exerts little or no influence.

Second-ary phenomena. Headache, languor, depression, vertigo, vomiting, epileptiform states. Peculiar dyspnea, apnea, stupor, fatal coma.

Acetonemia of intestinal origin is by some considered to be an affection sui generis. Whether such an acetonemia is the cause, or the concomitant or the effect of a certain symptom-complex with which the pediatricist is confronted, is a question that must be decided in each and every instance. Personally, I believe that this acetonemia may sometimes be the etiological factor, at other times an associate phenomenon of a syndrome, and still at other times the result of the pathologic process underlying the entire disturbance.

By intestinal acetonemia, which, with less justification, but for convenience sake, may also be called intestinal acidosis, I understand a condition in which ketones and allied bodies in a preformed state have, on account of a deficiency on the part of the liver, found their way into the general blood stream. These ketones, their associates and progenitors are rather innocuous in themselves. They are elaborated in the intestines and may act therein as local irritants. In small amounts they are probably of physiologic occurrence, and the presence of traces of acetone in the urine may be and has been considered a normal phenomenon. It is their overproduction in the intestine with which we have to deal at this moment. The ketones are very readily absorbed from the alimentary

tract. In case comparatively large amounts of these substances are quickly taken up and carried to the portal circulation, the autoprotection of the organism against enterogenous substances, be they poisonous or excessive in quantity, may fail. The absolutely healthy organism is equipped to dispose of these products by various means, no matter whether they are the result of normal or pathologic processes.

First.—Some enterogenous substances—prior to absorption—leave the body in the gaseous form at the distal ends of the alimentary canal, others are excreted with the feces.

Second.—A number of intestinal substances are converted into innocuous material, in which form they are either transmitted to the circulation or are excreted with the feces.

Third.—The normally functioning liver prevents from entering the general circulation or transforms into innocuous compounds such enterogenous (toxic) material which has traversed the intestinal mucosa and found its way into the portal circulation.

Fourth.—The antibodies circulating in the blood may cause attenuation or inactivity of the toxic matters conveyed to the blood.

The last mentioned autoprotective means of the organism is without effect as far as the ketones are concerned. On the hand of the other three possible modes of autoprotection, it is clearly evinced that in the last instance no intestinal substance can enter the general circulation when the liver function is efficient and faultless.

The first autoprotective eventuality, namely, that an enterogenous substance may leave the body in a gaseous form at the distal ends of the alimentary tract, or may be excreted with the feces, certainly seems to apply to the incompletely or improperly converted fatty acid products. The ketones, particularly in infants and young children, may be exhaled by the mouth and may be expelled together with the intestinal gases or the feces. When contained in the expired air, the ketones are probably of catabolic origin in the majority of the cases; when the odor of the combined ketones comes from the mouth while the patient holds his breath, these may have been derived from the alimentary canal, and this is doubtlessly the case when the ketones are contained in the flatus or the feces alone. By the direct expulsion of these substances from

the gastrointestinal tract the body protects itself against their possible local and general influences.

The second autoprotective measure, according to which certain intestinal substances are converted into harmless material, in which form they are either transmitted to the circulation or are excreted with the feces, is also of importance as regards the great bulk of low fatty acids that are evolved during normal digestion, or which are rapidly produced when certain nutriments are ingested or during certain pathologic processes implicating especially the gastrointestinal apparatus.

The fatty acids of low molecular weight are normally, to some extent, transformed into gases like CO_2 , N_2 , CH_4 , and H_2 ; the remaining acids, neutralized by fixed and volatile alkali, are absorbed, after which they undergo oxidation.

Very little of a definite nature is known of the pathology of the low fatty acids that are produced enterogenously. These acids, though neutralizable, are neither emulsifiable nor saponifiable. About their fate in the organism, in case they should not have been rendered absorbable, we possess but very meager and vague data. We know that the volatile fatty acids are always present in the stools of infants with pronounced acid reaction, but the fecal acidity is never of such high degree as to account for the total low fatty acids that are generated in the alimentary tube of a milk-fed baby. (Besides, a certain proportion of the fecal acidity is undoubtedly caused by the intestinal micro-organisms whose number and activity are often pathologically increased in many of the alimentary disturbances of infantile life.) Moreover, it stands to reason that but a certain proportion of these acids, when ketonized, leave the body, partly in a gaseous form and partly combined with the feces. There is, hence, nothing left but to assume that while in the pertaining cases the low fatty acids remain in the bowel, where they combine with fixed and volatile alkali as long as there is a supply of these, and where they are split up in various ways, the volatile ketones, one of their disintegration products, for reason of their free and rapid diffusion, are in a measure enabled to enter the portal circulation. It is here, now, that the third and most important autoprotective reaction, that due to a well functioning liver, should be displayed, for this, be it

on account of a special property or on account of the mere presence of glycogen within its cells, accelerates the breaking down of the ketone bodies.

The liver, failing in its ketone-splitting function, can no longer prevent the transitory flooding of the blood stream with ketone substances. The alkali supply of the tissues and blood, so it appears, has nothing, or but very little, to do with the neutralization or disintegration of the ketone substances that already existed before they were transmitted to the blood current.

ACIDOSIS

By WALTER D. LUDLUM

Assistant Pediatrician, Kings County Hospital; Attending Physician, Kingston Avenue and Seaside Hospitals
Brooklyn—New York

Acidosis is a condition showing itself to be of great and constantly more obvious, if not greater, importance, and yet the word has been used variously and has not a distinct and universally known significance; therefore, a definition is in order; the following seems fair:

Acidosis is a condition demonstrated by the presence of acetone in excess and diacetic acid in the urine, thus suggesting their pre-existence in the blood and tissues. At the present time, it is not even known, so far as I know, whether these and allied substances are responsible for the symptoms seen or are mere indices and concomitants of the actual causes. At least, it can be said that these two substances and Beta-oxybutyric acid are present in such states of acidosis and are our easiest index to its occurrence.

What is the source of these bodies? Beta-oxybutyric acid is $\text{CH}_3\text{—CHOH—CH}_2\text{—COOH}$.

This oxidizes readily to: Diacetic acid, which is $\text{CH}_3\text{—CO—CH}_2\text{—COOH}$.

And this, in its turn, to: Acetone, which is $\text{CH}_3\text{—CO—CH}_3$.

Diacetic acid is made up of two molecules of acetic acid by dehydration, thus: $\text{CH}_3\text{—CO—}\boxed{\text{OH—H}}\text{—H}_2\text{C—COOH}$.

One might, and, indeed, must, go far more deeply into the chemistry to have a reasonable understanding of this subject, but this

is too abstruse for hasty and oral presentation and the above will suffice for our present purpose.

These acetone bodies, as they are often conveniently termed, may be formed from any of the three types of food materials: from the carbohydrates they seem not to come as a pathological fact; from the proteid materials, by way of the amino-acids, they may be and, probably, sometimes are, but their main source is apparently the fatty acids.

Acetone is a normal constituent of the urine, but in minute quantity and as an indication of acidosis it is present in excess; oxybutyric acid is an intermediate product in the catabolism of the fatty acids, while diacetic acid is an irregular and adventitious product. Whatever the real *cause* of acidosis, the presence of these bodies in the urine would at least indicate an inadequate oxidation of, usually, the fatty acids, by reason of a deficient supply of alkali. The condition occurs usually where there is an insufficient supply of carbohydrate or inability to utilize it, and this means a great variety of conditions such as malignant growths, starvation, post-operative, etc., but in children chiefly cyclic vomiting and other recurrent disorders.

If we accept as a definition of acidosis the mere presence of these bodies in the urine, it is interesting to note how often this takes place with apparently entire unimportance; it would be interesting to have frequent investigations made to learn how often they are found and what, if any, are the regular associations of their presence. One report of this kind I read with great interest, by Frew, in *The Lancet*. Summarized, it is as follows:

The urine of 662 unselected cases was examined; of these, 408 cases, or 61.6 per cent., showed acetone at some time; of these, 2 cases were diabetic coma; 150 (22.5 per cent.) were gastroenteric; 256 (38.5 per cent.) unaccounted for under ordinary headings.

The most constant time for the occurrence of the acetone was 36 hours after admission, and it was always gone by the fourth day.

The system of the body affected by the disease seemed to have no influence, whether gastroenteric, pulmonary, etc., nor did the particular disease, except that in typhoid it was very low, only 15.3 per cent.

The age incidence was interesting; acetone was found in children under two years in 47 per cent.; over two years in 68 per cent.; under one year of age it was found in 41 per cent. of the cases. The maximum was between three and four years, namely, 84 per cent.

Of 11 breast-fed babies put on cow's milk, all developed acetone, which cleared up in three days, while of 38 under one year bottle-fed only 6 (15 per cent.) did.

He called attention to the following observations: That acetonuria occurred *after* admission; that it was more frequent in children over 2 years with a change of diet in the direction of simplicity, and that it was not affected by the disease suffered.

He drew the following conclusions: That acetonuria is common in childhood; that it is due to carbohydrate starvation, usually caused by failure of digestion, not by lack of supply; that this loss of digestive capacity may be due merely to change of diet; that it is more easily caused the younger the child; that three days are required for the accommodation of digestion; that disease has little effect.

This paper was not designed to be a detailed presentation of the theory of acidosis, but merely to emphasize it as a subject of practical importance and to intimate again, perhaps, that often that is found which is sought. This practical part may occupy small space as compared with this introduction, intended only to lead up to it.

I shall not give detailed histories of cases, but merely use some as illustrations of the points which seem to me of interest and importance.

A girl of four, typical cyclic vomiting, nothing unusual in the attacks, diagnosis long made, attacks occurring irregularly, but at intervals generally between six and twelve weeks; child always thin, never a hearty appetite, attacks moderately severe, lasting about three days with vomiting very frequent. For four weeks a fairly close regimen had been carried out; then she went out of town and an attack promptly followed. What was the cause? Change and excitement? At least, it is to be observed that attacks are frequently thus precipitated. On strict diet and other

treatment she has had only one mild attack in six months, but she does not grow fat, not even decently.

A baby of ten months, nursed four months, fed since mostly on malt soup mixtures by one of the most eminent pediatricists; growth slow, weighing $16\frac{3}{4}$ pounds from a birth weight of 8 pounds. Home from the country a week, no change in formula or the milk. Patient has been vomiting two days, bowels were mildly disturbed the day before being first seen, but were perfectly good that day; temperature, 101 deg. F.; presumptive diagnosis, indigestion. Vomiting continues when on barley water only, not very frequent and violent, but enough; bowels good. Acetone and diacetic acid were found; diagnosis: recurrent or acidemic vomiting. The return to normal diet was very slow.

A girl of four years, sick two days with a little fever and a mild sore throat, but with persistent vomiting for thirty-six hours; no history of a previous attack of such vomiting. Examination shows a definite follicular tonsillitis, clean tongue, temperature of 101 deg. F., no other abnormality on direct physical examination. I suggested to the attending physician that, if it were not for the tonsillitis, I should call it cyclic or acidemic vomiting; acetone and diacetic acid were found, alkaline treatment instituted and the condition subsided in twelve hours.

A boy of four years, under my own care from birth; practically always well and well nourished. One attack, a year ago, of bronchitis with little fever and an abnormal amount of vomiting. Six weeks ago bronchitis with persistent vomiting all the first day; bronchitis severe, diffuse, moist, but after two days taking on a mildly asthmatic character, scarcely any fever. Now another attack of bronchitis coming on very suddenly, from being nearly well he is prostrated in a couple of hours, looks seriously sick, temperature only 100 deg. F., diffuse râles over one lung, few in the other, in the "full" lung they are moist, some of the other are asthmatic. Acetone and diacetic acid are present; main treatment is alkaline.

When we find recurring attacks of any condition in a child from two to ten years of age, less often below or above those ages, especially if it be recurrent vomiting or bronchitis, and particularly if the latter has an asthmatic character, it would be worth while to look for acetone and diacetic acid.

If we find persistent vomiting even without a history of previous similar attacks, without adequate cause, with normal temperature or slight elevation, with prostration disproportionate to the other symptoms, even if there be present some definite lesion as tonsillitis or bronchitis, an acidosis may be suspected and sought.

Neither from observation nor reading have I been strongly impressed with the idea that acidosis was an infection toxicosis, though this is suggested by several. It seems, without question, a metabolic disorder due to inherent predisposition brought out by diet unsuited to that predisposed capacity, with perhaps—or occasionally—an incidental disease.

On this basis the treatment is as follows: With all due regard to the fact that all kinds of food may be the origin of these acetone bodies, they seem to come usually from fat, especially butter-fat; occasionally from proteid. Sugar seems to facilitate their formation, while starch inhibits it. Therefore, a diet from which cream is eliminated, meat is greatly reduced and sugar reduced to a minimum, serves to prevent the condition. The chief difficulty with this is that so little is left, and I confess to the trouble I find in keeping these youngsters fattened. Having cut his fare to this minimum diet, one should carefully try out increases and endeavor to learn the specific capacity of the individual case.

Medicinally, the treatment lies in alkaline medication; bicarbonate of soda typically or sodium or potassium citrate with at times salicylates. These measures seem to work. It is almost superfluous to mention the virtues of fresh air, baths, rest and exercise properly adjusted with emphasis on the rest, climate and all hygienic measures; lack of excitement and undue activity is important.

Not to speak of them as conclusions, we might make the following suggestions:

That acidosis, or acetonemia, is a common condition in childhood.

That the transient presence of acetone in the urine is often unimportant, but yet definitely significant; at other times it is both important and significant.

That acidosis occurs in childhood most often in conditions with a tendency to recurrence.

That it will be instructive and interesting to watch for it and find as fully as possible its associations and significance.

A CLINICAL STUDY OF A CASE OF ACIDOSIS

By GEORGE F. LITTLE

Assistant Clinical Professor of Pediatrics, Long Island College Hospital;
Pediatrician, Kings County Hospital; Consulting Pediatrician,
Mercy Hospital, Hempstead, Long Island, and the
Brooklyn Children's Aid Society

Brooklyn—New York

This child has been under my constant care and supervision since birth, and an excellent opportunity has thereby been afforded for the study of the manifestations of acidosis.

Boy—four years and nine months of age.

Past History.—Grandparents negative. Parents alive and well. No inherited disease or dyscrasia. Prolonged labor, some sixty hours before sufficient dilatation was secured to permit rotation by forceps from an occiput posterior position. Owing to shock, breast milk was insufficient during the first ten days, then failed. Through nutrition difficulties, in intensely hot weather, the infant showed a considerable degree of malnutrition, but was brought to normal condition in the first three months.

There have been no illnesses of note, except pertussis. Surroundings, as to care, diet and hygiene, have been ideal. The bowels have been regular.

Present Condition.—Robust health. Height, three feet, eight and a quarter inches. Weight, fifty pounds, two ounces. Some two and a half inches and nine pounds above the average for boys at five years. The child is active and muscular, with an unusually bright mentality; he is of nervous temperament.

History of Acidosis.—At about two years of age, there was an attack of uncontrollable vomiting, with rise of temperature, lasting some six hours. At the end of twenty-four hours, nourishment was retained, and the little patient was as well as ever in three days. At the inception of this illness, dietary errors were probed for, but could not be found. There had been two previous attacks of similar nature, at intervals approximating two months, without discoverable etiological factors. The so-called cyclic, recurrent or periodic vomiting was, therefore, suspected and a specimen of urine was secured in the first day of this second recurrence. There was a showing of diacetic acid and acetone.

For the succeeding two years there were vomiting attacks, in cyclic form, the intervals approximating two months, except that frequency was increased during pertussis, which lasted through the summer of 1913. Beginning with the summer season of last year, the periodic disturbance has become more frequent, in spite of treatment; intermissions for some time being about three weeks, and once but two weeks. Recently the attacks are seen four to five weeks apart. With this change in frequency there has been a change in symptomatology in most of the recurrences.

Character of the Usual Attacks.—Until the later period just mentioned, the prodromal symptom was nausea. This was shown, in younger days, by partial refusal of food at one meal, entire refusal at the next, if offered. The little patient would state that he "felt sick in his throat." Later, with increasing powers of observation, the hand would be placed over the epigastric region to locate the place of sickness. After a somewhat varying time—more often ten or twelve hours after the first sign of nausea—active and uncontrollable vomiting supervened; this manifestation lasting around six hours. The stomach was emptied in the first couple of efforts, but, for perhaps two hours, further efforts at emesis were made at intervals of about ten minutes, productive only of small quantities of bile-like fluid. Periods of rest, in the succeeding few hours, gradually lengthened—fifteen, twenty, thirty minutes—with cessation of vomiting in the average time limit mentioned. Nourishment, commenced in very small quantities, could usually be retained about twenty-four hours after the initial nausea. Fever was regularly present, mainly during the first day—101 deg. to 104 deg. F. The patient was normal again in activities around three days from the beginning of the illness—except that the appearance showed, and the scales proved, a loss of weight of several pounds. This was generally regained in a week. Preceding an attack the bowels have usually been somewhat loose for a day. After the acute symptoms there is constipation for several days.

It is interesting to note that, in all the past history, the prodromal nausea has invariably been followed by active vomiting, except that in the last three months there have been two occasions where the nausea has been followed only by slight malaise, for a day or two, where the major attack, in other words, seem to have aborted.

A Change in the Picture.—That the symptoms preceding stomach involvement may, in some cases, be of a respiratory type is recognized—coryza or asthmatic manifestations appearing. I have not, however, met with a case, in practice, or in literature, where the development of these symptoms has been so alarming as in two or three of the recent attacks in the child under discussion.

Early in the summer of 1914 there was the onset of what was apparently an ordinary cold—rhinitis one day, bronchitis the next. The patient was restless and feverish during the third night, with some acceleration of respiration. In the morning the general picture gave an opportunity for figuring that the bronchitis had rather suddenly invaded the small tubes and probably had reached the alveoli. The temperature was 103 deg. F., the pulse 140, respirations 55, shallow, labored, irregular. Cyanosis showed on the face and under the finger nails. There was marked dyspnea, with recession of the chest wall, above the clavicles, on inspiration. The physical signs were indefinite. Active measures of treatment were instituted for relief of congestion—full catharsis, steam inhalations every two hours, mustard jackets, covering the whole thorax, every six hours.

The condition maintained, practically unchanged, throughout the day and early evening; there was then a change for the better, the respirations falling to 35 within an hour, with increase in depth and regularity. Vomiting set in for the usual period. The child was convalescing in the morning, with the respiratory tract in normal condition. The urine showed acetone.

An attack, of similar nature to the above, showed itself several weeks later, but was somewhat milder in its manifestations. Early in October, a recurrence of the same type supervened with symptoms, on the part of the respiratory tract, more alarming than those first noted. Following a coryza and a laryngitis, the boy for thirty-six hours presented the picture of an overwhelming pneumonia. Temperature 104-104.5 deg. F., respiration averaging 60—urgent dyspnea and cyanosis. Dr. Thomas R. French and Dr. Elias H. Bartley saw the case with me; both admitted the picture to be one of pneumonia, rather than of asthma, although the numerous râles of all kinds in the chest were more indicative to Dr.

Bartley of the latter condition. At the end of the period spoken of, the respiratory symptoms cleared up, practically within an hour, and a period of vomiting supervened.

A month later, the child showed a coryza, followed by a laryngitis and then by a frank asthma, of mild type, and of only a few hours' duration—followed again by the vomiting.

Attacks since this time have been preceded by the coryza and laryngitis, but have been free from other respiratory symptoms—except that the most recent recurrence assumed the old type, with absence of any respiratory involvement. It is notable that, while intervals have been lessened of late, the severity of the gastric crisis has been modified, there perhaps being only some half dozen vomiting spells, extending over a period still approximating six hours.

Treatment During Attacks.—At the first suspicion of onset, I have given one and a half to two grains of calomel, followed in a few hours by a tablespoonful of the milk of magnesia—this latter has often been refused on account of increased nausea, and if forced would not be retained. In the recent periods, where nausea has been preceded for a couple of days by respiratory involvement, it is possible to push alkalis. Twenty grains of sodium bicarbonate, Squibb, are given, well diluted, at hourly intervals. As it is naturally inadvisable to alkalinize the gastric juice during stomach digestion, perhaps half a dozen of these doses can be exhibited in a day.

The little patient is given ice-water, or preferably seltzer, in small quantities, but very frequently, during the vomiting, for thirst is pressing and even though the liquid be not long retained, it serves in a measure to wash out the stomach and makes emesis easier. When the seltzer is not objected to, a little soda is added. Small pieces of ice in the mouth are grateful; in younger years the ice was wrapped in a piece of gauze, the distal ends held by the nurse. In several of the sharp and more prolonged attacks, soda solution, one drachm in four ounces of water, has been placed high in the rectum, at four-hour intervals. Hypodermoclysis has not been found necessary.

When emesis ceases, and it is found that a tablespoonful of water is several times retained, an equal quantity of skimmed milk is offered—this has been peptonized by the warm process for ten min-

utes and is given ice-cold at half hour intervals. Toleration being shown by a few feedings, the quantity is doubled for several occasions, then for several more two ounces an hour are offered—followed by four ounces at two-hour intervals. Zwieback, or a cracker, light cereal gruels, a little vanilla ice cream, soft egg and toast are added to the diet, as indicated, in the next couple of days, and in the order mentioned. One-third to one-half of the cream is added to the skim milk on the second day and peptonization somewhat reduced. Cold peptonization suffices on the third day, with two-thirds, or full, cream. Return to full diet is customary on the fourth day, sometimes a little earlier in mild attacks.

Interval Treatment.—Fats and sugars, in the diet of the boy, have been reduced as much as is consonant with physical demands for sustenance and growth. Regularity of the bowels is insisted upon—if a day passes without a movement, an enema is given at bedtime, or a glycerin suppository inserted—a laxative by mouth is exhibited at the same time; milk of magnesia, a tablespoonful, or phenolphthalein, grains one and one half. This latter drug is put up, by one of the manufacturers, in chocolate tablets, of this strength. These may be divided for younger children and crushed—the older child eats them with avidity.

As for medication, sodium bicarbonate is given twice a day in ten-grain doses. The taste of this, in solution, is likely to be objected to at first, but the distaste is soon overcome. The drug may be given in seltzer or vichy, which entirely disguises the flavor. A few days' intermission is allowed now and then. The solution is given between meals.

In accordance with a suggestion of Kerley,* sodium salicylate, from *ol. gaultheria*, was alternated, for several months, with the bicarbonate. Four grains of the former drug was given twice a day, in solution, with a little peppermint water added, for a period of five days. The alkali following for ten days. In this case, no beneficial results were noted.

All general hygienic measures for the maintenance of good physical condition are faithfully observed.

*Practice of Pediatrics.

A SUMMARY OF THE ESSENTIAL POINTS IN THE
DIAGNOSIS OF GASTRIC ULCER

By FRANK SMITHIES

Gastroenterologist, Augustana Hospital; Former Gastroenterologist, Mayo
Clinic, Rochester, Minn.
Chicago, Ill.

I. FACTS DETERMINED FROM HISTORY

(a) Frequently dietetic or hygienic irregularities. Males are more frequently affected than are females.

(b) History of recurring acute infections (la grippe—tonsillitis, exanthemata, etc.). Seasonal relation of distress not uncommon, exacerbations occurring in fall or spring.

(c) Association with disease of appendix or gall-bladder (with which ulcer, especially in subjects below 30 years of age, is often confused).

(d) *Periodicity of complaint*; occurs in from 75 to 85 per cent. of instances until complications set in. Between "spells" or "attacks" of indigestion, so called, there is generally good gastric health. Weight is not infrequently lost during attacks, and rapidly gained when such cease.

(e) *Epigastric distress*; present in more than 95 per cent. of instances. Varies in severity from discomfort to severe, gnawing or cramp-like pains. Discomfort has point of maximum location, subjectively, in practically 3 out of 4 cases. Pain has usually reached its height within four hours following meals. Pain comes on sooner *post cibo* in ulcers located near the cardia than where such are well toward the pylorus.

(f) *Food relief of distress* occurs in four out of five instances of peptic ulcer of the uncomplicated type. Relief of pain frequently bears relation to amount of food taken, i.e., a large meal gives longer relief than a small one. Pain is also relieved by vomiting, the taking of alkalies, by rest, diet and opiates.

(g) Vomiting occurs in more than two-thirds of instances; vomitus usually comes on at the height of gastric distress and when acidity is highest. Vomitus of food that has lain in the stomach longer than six hours ("delayed vomit") increases as complications (stenoses or perforation) develop. Pyrosis, water-brash, eructations and sour belching are common on ordinary diet.

(h) *Hemorrhage* (hematemesis or melena) occurs in from 30

to 40 per cent. of instances. While hematemesis is more frequent than melena, yet melena alone may occur wholly irrespective of the location of the gastric ulcer. Severe hemorrhage is accompanied by sign of shock and collapse.

II. FACTS ELICITED UPON PHYSICAL EXAMINATION

(a) Patient usually well nourished without toxic or cachectic appearance, unless pyloric stenosis or "hour glass" contraction has occurred.

(b) *Area of epigastric tenderness* in region of pyloric half of stomach. This is usually in the mid-epigastrium, but not necessarily so. The area is most frequently definitely local where acute or chronic perforation has taken place. A tender ridge may sometimes be palpated where a large chronic ulcer exists.

(c) *Dilated, splashy stomach* occurs where marked pyloric spasm exists or stenosis has taken place. If this is excessive, visible peristalsis (and rarely "reverse" peristalsis) may be noted.

III. FACTS ELICITED BY LABORATORY EXAMINATION

(a) *Test meal*—motility interfered with in more than 50 per cent. of instances. *Gastric acidity* increased as regards free hydrochloric acid in the majority of non-stenosing ulcers. In stenosing ulcers, with dilatation of the stomach, while free hydrochloric acidity may not be much above normal, the total acidity is increased in greater ratio. Blood may or may not be present in gastric extracts (macroscopically or by chemical test). Lactic acid is a rare finding. Pepsin and rennin are frequently increased.

Microscopically where gastric dilatation has occurred, fermentative changes are proved by the finding of great numbers of budding yeasts and of sarcinae (large and small types), together with remnants of retained food.

(b) *Stool*—may show nothing pathologic. Recent hemorrhages generally result in the passage of "tarry" stools for several succeeding days. Perforation of an ulcer to the pancreas not infrequently results in pancreatic inefficiency with passage of stools containing undigested food or ferment abnormalities.

During periods of the ulcer's activity, if the patient is kept upon meat-free or milk diet for several days, at the end of such time the stool may be shown, by chemical tests (benzidin or

guaiac), to contain blood. Progressive ulcers or ulcers undergoing cancerous change generally show blood constantly in the stools by chemic tests.

(c) *X-ray findings.* In many instances of uncomplicated ulcer no facts are returned after most careful examination by both fluoroscopic or plate methods. Complicated ulcers (stenosing, calloused, hour-glass-producing, perforating, etc.) are recognizable in nearly three out of four instances by the combined screen and plate methods.

A dependable clinical technic is as follows:

Empty the gastrointestinal canal by the administration of 2 ounces of castor oil in beer or malt extract.

Give a "*Motor opaque meal*," consisting of 2 to 4 ounces of barium sulphate (pure) or of bismuth subcarbonate in 6 to 8 ounces of cream of wheat, oatmeal, wheaten, or the like, at 4 A.M.

Six hours following examine, by means of the fluoroscopic screen, to locate the position of the motor meal. The presence of the opaque mixture in the stomach usually indicates anatomic interference with the onward progress of the food or furnishes evidences of gastric atony. Plates may be made at this time for purposes of recording the position of the motor meal.

A *second meal*, for purposes of studying gastroduodenal contour and activity, is next given. This consists of 2 to 4 ounces of barium sulphate or of bismuth subcarbonate in 16 to 24 ounces of butter-milk, fermillac or potato purée.

While the patient is taking this second meal the stomach is observed by means of the fluoroscope. Palpation is carried on and the patient examined in various positions. If suspicious contractions, peristaltic waves or other abnormalities appear, the patient should be reexamined on several successive days, before and after the administration of such antispasmodics as atropin (gr. 1/50 hypodermically) or tr. belladonna (gtt. xv every 3 hours for a day). This procedure aids in demonstrating the constancy or the transience of a local sign. Plates (frequently taken with the patient in different positions) may be next made for purposes of leisure study or for permanent record of positive or negative results.

When present, briefly, the *X-ray findings in gastric ulcer* are:

I. Positive signs. The "niche" or "accessory cavity" indicating calloused, penetrating ulcer.

II. Corroborative signs. (a) "Incisura," i.e., local evidence of halting of peristaltic rhythm by spastic contraction of circular muscle fibers in the vicinity of an ulcer. Best brought out on screen examination during or after palpation.

(b) "Hour-glass" stomach (bi-loculation). This may be permanent (callous ulcer, perforation, adhesion) or transient (local spasm, with or without ulcer), and should always be proved by repeated examination, with and without an antispasmodic (atropin, belladonna).

(c) Gastric residue—this may vary in amount. Its constant demonstration after six hours means atony or stenosis. Intermittently it may result from extra gastric or gastric pathology causing pyloric spasm. (cholecystitis, appendicitis, etc.)

(d) Fixation of all or part of the stomach (perforation, adhesion, fistula.)

(e) Area of tenderness to palpation usually localized at some part of the stomach shadow. Should always be checked by repeated examination before and after an antispasmodic.

(f) Alterations in gastric peristalsis, e.g., exaggerated peristalsis, intermittent, frequently associated with spasmodic closure and relaxation of the pylorus. Antiperistalsis may be seen on rare occasions.

THE DIAGNOSIS AND CLASSIFICATION OF DIFFICULT FEEDING CASES AFTER THE FIRST YEAR

By GODFREY R. PISEK

Professor of Pediatrics, New York Post-Graduate Medical School and Hospital; Professor of Pediatrics, University of Vermont
College of Medicine

New York

Numerous and worthy articles have been written with the object of making the subject of infant feeding simpler, and describing how to deal with those pathological infants known as "difficult feeding cases"; but comparatively little has been said regarding the diagnosis and management of patients in the early years of life who, although free from distinct constitutional diseases, suffer from marked malnutrition.

These children are brought to the physician because they are not

as robust as their neighbor's child of the same age; because they tire easily, because they are under-size, thin and pale. The mother complains that the child is extremely difficult to feed, capricious, or that the food "seems to do the child no good."

On examination they are found to be as a type, mentally well developed but physically much below the average; the musculature is flabby, the chest long and the thoracic capacity much diminished, the spinal muscles are relaxed, producing a poor posture. They are invariably anemic owing to poor oxygenation and constipation. They prefer to play alone and shun the active amusements of other children. The mother further relates that she has tried to force the child to eat and grow fat, but has not succeeded. Her physician has prescribed "tonics" but without success.

The family physician is just as capable as the pediatricist (to whom they are so often sent) to care for this type of child. How this may be done will be indicated in this paper, the object of which is to call to your attention a neglected field of practice in which recovery is dependent upon the conversion of food elements into blood and healthy tissue, and in which stress is laid upon practical hygiene rather than upon the use of drugs. Particularly must we make this plea for the child who has a *right* to begin his life work with a healthy mind in a healthy body.

The laity are beginning to appreciate that it is wise economy to correct minor ills by periodical examinations and systematic feeding regulations, rather than to await the occurrence of serious illness. Fortunately, these cases of late malnutrition are less frequently seen than the atrophic or marasmic artificially-fed babies in infancy; but when they do occur they often need more study and detailed management to attain success than the infants.

In infancy our difficulties mainly arise when we attempt to substitute artificial food for the human product, and when the attempt is made to fit the baby to the food, instead of adapting it to the delicate developing digestive system.

At a later period, when the teeth have erupted and the child has progressed to the point of taking semi-solid foods, there may occur as a result of faulty feeding, malnutrition, or even such profound changes as to put the child's life in jeopardy unless by skilled dietetic management the vital spark is fanned into life.

The analysis of our case records enables us to group the cases under the following captions: Those that have been difficult feeding

cases in infancy, and are still sufferers from nutritional disturbances which prevent them from assimilating foods ordinarily found in a dietary suitable to their age.

Those that have been fed on an unbalanced ration made up mostly of carbohydrates.

Those with an inherent intolerance for proteins or fats.

Another type is the child that has made fairly normal progress in infancy, but who later in childhood remains stationary or slowly loses weight and vitality. Here the condition often results from insidious dietetic errors. They are said to have idiosyncrasies to certain articles of food, which prove to be not so much idiosyncrasies of the child as of the mother; or the whims and fancies of the child are unduly considered until the dietary is extremely limited. They are said to have a poor appetite and, therefore, this jaded appetite is stimulated by misguided, misdirected efforts at meal time of story telling or amusements. Such cases mainly appear among the neuro-pathic children of the well-to-do, and among the city dwellers where outdoor exercise is restricted, but whose access to improper foods is proportionately great. Even among the poor coming to our hospitals the cause is very rarely due to insufficient food, but it is due to a poor quality of food, poorly selected, and still more important poorly prepared.

A close study of the antecedent feeding history, the present dietary and an intimate knowledge of the daily life is essential for proper future management.

The physician must inquire not only as to what the child is offered in his dietary, but what is the child actually getting. This is best reached by recording the likes and dislikes of the particular child and the average amounts taken.

Physical examination will disclose the loss of weight, the poorly developed body, flabby musculature, enfeebled heart and blood, dry skin and toneless abdomen, of this victim of malnutrition.

The weight should be considered in relation to the birth weight and the highest weight ever attained.

Examinations of the stools and of the urine are to be made not only once but on repeated occasions so that the measure of digestion may be obtained, and in order that we may determine what constituents of the diet were not used up. As pointed out by the writer in a

previous communication there is no intricate or complicated process necessary for a practical examination of the stools to determine suitable treatment.

The children under consideration will show no evidences in the stools or urine of disease conditions, but they will exhibit marked changes in their ability to assimilate the various food constituents. It may be well here to review some of these characteristics. In this we will also follow the lead of Morse and Talbot, who have made extensive studies of the child's stools. Grossly we may find large undigested food masses; the result of imperfect food mastication or of food so well comminuted, that the child bolted it without the need of chewing.

A simple test with litmus paper will give some evidence of the mal-assimilation of the proteins or the fats. In the former instance of protein putrefaction, a marked alkaline reaction is obtained, besides the distinct putrefactive odor; while on the other hand an acid reaction appears in fat and starch disturbances, accompanied by a butyric odor if the fats are at fault, or a sour lactic acid odor if it is the carbohydrates that are causing the trouble.

With the microscope we may further determine by finding an excess of fatty acids and soaps that the digestion is *normal*, but that assimilation is *abnormal*.

Fortunately, we are also able to elicit much information regarding the development of the intestinal tract by means of the X-rays; serial röntgenograms have paved the way to a closer understanding of the pathological conditions which obtain in many of the cases of marked malnutrition.

A dilated stomach with or without a sluggish atonic intestinal tract is found in many of these children, the condition usually being proportionate to the degree of malnutrition present.

It is not our purpose to discuss the treatment here. Suffice it to say, that the diagnosis once made and the type determined, the cure is dependent upon a knowledge of the principles of nutrition and the preparation of foods coupled with a thorough study of each case individually. Resourcefulness and a knowledge of the preparation of food is imperative. Food must be supplied that is agreeable, easily digested, and that still contains the elements essential for growth and development.

TUBERCLE BACILLI IN STOMACH CONTENTS

BY I. H. LEVY AND J. L. KANTOR

Syracuse, N. Y.

Recently, while examining the gastric contents of a tuberculous patient, one of us was struck by the resemblance of the extracted material to tuberculous sputum. A smear revealed the presence of tubercle bacilli. Since that time we have succeeded in demonstrating the organisms in this fashion in two other cases.

As the bacilli in the stomach come from swallowed sputum, it would seem best, for obvious reasons, to aspirate early in the morning, while the patient is fasting, and as soon as possible after arising. On the other hand, the ready demonstration of the organisms in the presence of food (as in one of our cases) would suggest the value of examining any available specimen of gastric contents in tuberculous suspects. There should be no difficulty in obtaining some contents for examination in every case, provided an aspirating device is used. Our own technic is to use a short tube with glass connecting-piece and an Ewald bulb. The bacilli are easily and beautifully stained by the ordinary Ziehl-Neelsen procedure. We have been able to show experimentally that the staining qualities of the organisms are not affected by HCl-pepsin digestion for periods corresponding to the retention of substances in the stomach, and that the bacilli can be demonstrated after even a month's digestion in vitro, provided the staining time is appropriately lengthened (15 minutes steaming, one-half hour in cold). We have occasionally seen a large, plump, acid-fast cocco-bacillus in the stomach contents—possibly similar to the organism described by Smithies*—but this bears no resemblance whatever to the bacillus of tuberculosis, and should cause no confusion in diagnosis.

It is not, of course, in ordinary cases of pulmonary tuberculosis that the method herein described can lay claim to any practical value as a diagnostic measure. Nevertheless, there seems to be a fairly important group of conditions in which expectorated sputum is unavailable and where such a method should be of service. The following applications suggest themselves: (1) In tuberculosis of infants and young children, where this method should give results

*Smithies.—*Am. Jour. Med. Sci.*, Feb., 1915, CXLIX, p. 193.

at least as good as the throat-tickling procedure now generally advocated; (2) in incipient stages of tuberculosis, where expectoration is absent, and where it is inconstant; (3) in cases of miliary tuberculosis to supplement the search for the bacilli in the urine and stools; (4) in all unconscious states, and in tuberculous meningitis to supplement the search in cerebrospinal fluid; (5) in tuberculosis in the insane.

SUMMARY AND CONCLUSIONS

1. We have been able to demonstrate tubercle bacilli in the stomach contents of three patients suffering from pulmonary tuberculosis.

2. We believe that where expectorated sputum is unavailable (as in the group of conditions mentioned above) a certain number of positive diagnoses of pulmonary tuberculosis can be made with the aid of the stomach tube, as already described.

3. We believe that an early positive diagnosis of pulmonary tuberculosis can be made *more frequently* by the gastroenterologist, should he adopt the custom of searching the fasting contents for tubercle bacilli in all cases suspected of suffering from the dyspepsia of phthisis.

AUSCULTATION AT THE ACROMION PROCESS

(MEMORANDUM AND REJOINDER)

By ROBERT ABRAHAMS

Adjunct Professor of Medicine, New York Post-Graduate Medical School
and Hospital; Consulting Physician, Manhattan State Hospital
and Home of Daughters of Jacob
New York

Since the publication of my paper on the subject of "Auscultation at the Acromion Process, its Significance in Apical Disease" (ARCHIVES OF DIAGNOSIS, April, 1913) I had the extreme satisfaction to learn of the indorsement of my views by many workers in the field of pulmonary tuberculosis. I also had occasion to demonstrate this special form of auscultation to hundreds of physicians, from all corners of the country, who come to the New York Post-Graduate Medical School to brush up the old and ring in the new of things medical. And while there were some who came to scoff, eventually

all remained to pray. The ease with which, even the uninitiated, acquire the art of auscultating the acromion ends of the clavicles is one of the best arguments in favor of its practice.

Yet once in a while a voice or an echo is heard in opposition to this method. In order to satisfy the honest opposition, a short review of the subject is necessary.

The trouble with those who find fault with the method is that they lay stress exclusively on the modified and amplified respiratory sounds which are heard over the acromion processes. As a matter of fact, auscultation at the ends of the clavicles will bring out moist, dry and musical râles which are not obtained by direct examination of the apices. Friction sounds are extremely rare over the apices in the early stages of tuberculous infiltration, but are comparatively frequently heard over the acromion processes. A little practice will enable one to exclude muscle sound. Strong coughing and forced breathing may bring out mucous râles over the ends of the clavicles, but will not over the apices. Now, those gentlemen who honored me with their adverse criticisms have utterly failed to mention these immensely superior signs of early tuberculosis involving the upper parts of the lungs.

Perhaps it may be well to place the normal auscultatory sounds of the apices in parallel columns:

RIGHT APEX	LEFT APEX
1 broncho-vesicular breathing.	1 vesicular breathing.
2 clear, well-defined spoken voice.	2 ill-defined spoken voice.
3 clear and distinct whisper sound.	3 muffled and indistinct whisper sound.

Now whoever approaches the acromion process should think of these physiological differences between the two apices. He should also bear in mind the difference between vesicular breathing and broncho-vesicular breathing, namely, in the first, inspiration is heard from the beginning to the end, while expiration is only half or a third of inspiration; in the second, both inspiration and expiration are of

the same duration. In health, these qualities of the respiratory murmurs of the respective apices are preserved and continued over the acromion processes, with the very important addition that they are amplified, one or two degrees. An exception is often found in the left acromion process, where the expiratory sound is more pronounced than the inspiratory.

The spoken voice and the whispered sound are equally modified and amplified to a degree which would be classed abnormal when heard over the apices.

Now what happens in disease of the apices? Auscultation over the left acromion elicits marked broncho-vesicular breathing; louder spoken voice and very pronounced whispered sound.

Auscultation over the right apex yields almost tubular breathing with distinctly prolonged expiratory sound and unquestionable whispered pectoriloquy.

While these auscultatory phenomena are unmistakably apprehended over the acromions, one may at the same time hear very little *auscultatory* changes by direct auscultation of the apices. And herein is the crux and significance of auscultation of the acromion processes in early tuberculosis of the apices.

But our good friend and careful investigator, Dr. Joseph H. Barach, says that "between the prominent findings at the acromion in the normal and the findings in the slightly or moderately diseased, I believe no one can differentiate with certainty and accuracy." (ARCHIVES OF DIAGNOSIS, July, 1914.) This statement is based upon an "I believe."

No one values the work that Dr. Barach did, of which I only learned lately, in his study of the sound conducting properties of the bones of the thorax more than I, yet I venture to tell him and others that the slavery of statistics is not conducive to the mastery of clinical facts. Live clinical *impressions*, gathered for years at large clinics where patients are carefully watched and results scrupulously noted, have in them a potential and manifest energy that the dry-as-dust statistics may never hope to possess.

We are told that "50 young men," who submitted to acromion auscultation, had chests "beyond a doubt perfectly healthy," yet they showed an amplified breathing at the acromion processes. If they did, "beyond a doubt" a good many of them had had tuberculosis of

the apices, which show and will continue to show auscultatory changes in the acromions and perhaps in the apices. For I convinced myself and others, that just as auscultation at the acromions may indicate active, so it may indicate passive, or healed, tuberculosis.

I have subjected quite a number of patients in whom acromion more than apical auscultation pointed to an early lesion to X-ray examination; in some the result was negative yet eventually showed unmistakable signs of tuberculosis; in others there were marked infiltrations, and the wonder was that so few auscultatory signs were obtained by direct examination of the apex, and in still others, by far the largest number, there was hyperemia or congestion of the apex, or as the radiographer would say, "the apex looked cloudy," but no ocular evidence of infiltration. For such cases, the incipiently incipient as it were, auscultation at the acromion processes is a great boon, a great help, a veritable discovery.

A FURTHER PLEA FOR ABRAHAMS' ACROMIAL AUSCULTATION IN THE DIAGNOSIS OF IN- CIPIENT APICAL TUBERCULOSIS

By NATHAN MAGIDA

Clinical Assistant in the New York Post-Graduate Medical School and Hospital
New York

I am glad to find new cause for bringing this subject once more before the profession. Dr. Joseph H. Barach, in the *ARCHIVES OF DIAGNOSIS* for June, 1914, contributed an article in which he reviewed my paper on "Acromial Breathing as an Aid in the Diagnosis of Incipient Apical Tuberculosis," which appeared in the *New York Medical Journal* of December 27, 1913, and his conclusions seem to be that we were too hasty in considering this sign of great value as represented therein.

Before I go any further, I beg to accord to Dr. Barach all credit due him for his work on "Bone Conduction of Sound." I must confess that when I wrote my article I had no knowledge of his study on the subject.

In the present paper, besides maintaining our position in this matter, I wish also to give somewhat fuller details in reference to the use of this sign.

It seems to me that Dr. Barach did not consider the matter at hand from our point of view, his contention being that "Acromial Breathing" may be elicited in normal cases; that is, the sounds heard over the apices are more clearly perceptible over the acromial ends of the clavicle. This is true, and is exactly the principle upon which we are working. But, neither Dr. Abrahams, in his original article, which appeared in the ARCHIVES OF DIAGNOSIS of April, 1913, nor I claim to make positive diagnoses of incipient apical tuberculosis whenever we get amplified auscultatory signs at the ends of the clavicles. What we do claim for "Acromial Breathing," however, is that it is an aid—and a valuable aid—in the diagnosis of incipient apical tuberculosis. This has been proved repeatedly to ourselves and also to many physicians taking courses in physical diagnosis under Dr. Abrahams at the Post-Graduate Hospital.

There is, to my mind, no *one* sign which can be put down as positively diagnostic of early tuberculosis. For this reason it does not seem to me amiss to be familiar with, and use, as many signs as possible in attempting to make a diagnosis of this disease in its very early stages. As I stated in my article, this sign is *constant* in incipient apical cases, and that is the reason for our ardent enthusiasm in the matter. Still, even though we constantly find it in these cases, that does not mean to imply that we base our diagnosis *on this sign only*. Hence, if, in examining a case, we find, for example, a change in percussion, and, on listening over the acromion end of the corresponding clavicle, auscultatory signs are marked, ample, exaggerated, though no such changes are apparent by direct examination of the apex, we think that we are justified in making a diagnosis of incipient tuberculosis. The only difference, of course, in examining the right apex is that we have to take into account the normal auscultatory differences between the right and left apices.

In further proof of the value of this sign in early cases, I wish to offer the results which we have obtained in a series of X-rays taken after we had made a diagnosis of incipient apical tuberculosis by, or with the aid of Abrahams' method. All these cases had so very few signs and symptoms that an appeal to the X-ray was deemed of value as a trial. It must also be kept in mind that the

changes in incipient apical tuberculosis are usually too slight to show tangible diagnostic signs on the radiographic field. Still, out of 15 cases, 8 were returned as positive, 3 doubtful, and 4 negative. The following are instances of the findings in positive cases:

1. "The pulmonic fields are of even size, but unevenly illuminated, there being a clouding of the entire left pulmonic field below the clavicle, and of the right above the clavicle. There are numerous small miliary deposits at the right apex, while the vascular markings of the left upper lobe are seen with unusual distinctness, and there are evidences of miliary deposits which are confluent in the left upper lobe. These findings indicate pulmonary tuberculosis in the first stage." Direct auscultation unsatisfactory.

2. "A radiographic examination of the chest discloses the presence of a diffuse tuberculous infiltration of both lungs. Both apices are clouded, and the left chest discloses more extensive lesions than the right." Acromion auscultation was most pathognomonic.

3. "A radiographic examination of the chest shows a diffused miliary tuberculosis of both lungs with partial infiltration of the right apex, wherein are visible numerous calcific foci. The hilum shadows are large. There is evidence of some dilatation of the bronchi at the root." Acromion auscultation told the tale.

4. "A radiographic examination of the chest shows evidence of infiltration of the left apex. The distribution of the lesion is perivascular." Acromion breathing and whispered sound suggested the diagnosis.

I hope that the above will clearly outline our position in this matter and that we shall no longer give the impression of making this one sign a positive one of tuberculosis by itself. All that we wish to claim for it, is that it is a very good aid—and helps to obtain signs which could not be obtained otherwise.

While on the subject, I should like again to bring out the points to be observed in order to get the full value of this method. The following has, no doubt, been pointed out by the other gentlemen who have written on this subject, but I do not think that it would be out of the way to review them:

1. The bell of the stethoscope used should not be of too large a circumference, as most of these patients are rather thin in the acromial region.

2. If the bell does not fit snugly over the acromion process so that all external sounds are excluded, the skin of the surrounding area should be pinched up on either side of the bell.

3. The patient should be instructed to breathe deeply and not noisily.

4. Examination of the nose should always be made before examining the acromion process, in fact, before examining the apices, because patients with nasal obstructions of any kind give exaggerated breathing sounds over the apices which are much more marked over the acromion processes.

5. The heart should always be examined before the apices, as mitral disease usually gives a certain amount of congestion in this region somewhat resembling incipient tuberculosis.

6. Most important of all, the apices should always be examined before the acromion processes, as this method is really a comparative one and its full value cannot be appreciated unless the examiner knows the normal sounds elicited over the apices.

I beg to thank Dr. Seth I. Hirsch, of the Post-Graduate X-ray Laboratory, for the radiosopic information.

THE EAR COMPLICATIONS IN INFLUENZA

By HAROLD HAYS

Assistant Otological Surgeon, New York Eye and Ear Infirmary; Assistant Laryngologist and Otologist, City Hospital, etc.

New York

During epidemics of influenza and during the seasonal period of the year when influenzal infections are more prevalent, ear complications are frequently seen. These are usually secondary to influenzal infections of the nose and throat, which often result in severe inflammatory reactions of the accessory sinuses of the nose at the same time.

It is seldom, however, that the infection in the ear is caused directly by the influenza bacillus. In the majority of instances, the ear condition arises from a general lowering of the resistance, which allows secondary infecting organisms to penetrate through the already infected mucous membranes of the eustachian tube. These organisms, in the order of their importance, are the strep-

tooccus mucosus capsulatus, the pneumococcus, the streptococcus pyogenes, the staphylococcus pyogenes aureus, and more rarely the bacillus coli communis, the bacillus proteus, and the Friedländer bacillus. Whether the influenza bacillus is really present in the ear infection, or whether it is merely overgrown by the other organisms, it is hard to say. The influenza bacillus is extremely difficult to grow except on blood media, and it is possible, therefore, that it may be present many times, but not discovered.

The ear complications of an influenza may be extremely severe, depending upon the virulence of the organisms and the resistance of the patient. In the majority of instances, the patient suffers from a dulness in the ears, perhaps with sharp shooting pains. The drum appears normal, but examination with the pharyngoscope shows a very intensely congested eustachian tube which completely closes off the middle ear. We thus have what is called an acute tubal catarrh, which, if taken in time, will resolve very nicely and result in no complications. The dulness in the ear is caused by a rarefaction of the contained air within the middle ear cavity.

The treatment of the eustachian tubal orifice at the time when the inflammatory condition is confined to these parts is extremely important; and it is possible in many instances to retard the inflammation, thus preventing an infection in the middle ear itself, by timely and proper attention to such parts. Inflation of the middle ear through a highly inflamed tube, the mouth of which contains many virulent organisms, is pernicious and unwarrantable. In such cases it is wiser to treat the cause of the condition and the inflamed mucosa of the nasopharynx rather than attempting to force a moderate amount of infected air into a part that is well closed off by Nature. Once the infection has been retarded, cautious inflation of the middle ear is necessary; and, fortunately, almost all such cases are cured even when the middle ear symptoms are ignored for weeks.

In some instances the infection creeps up these congested tubes, causing an acute otitis media with or without fluid. As I have just stated, this may be caused by untimely inflation. The ear drum becomes intensely engorged, but resolves in a great many cases when proper medication is given to the tube and when the ear itself is irrigated with hot saline solution. If fluid is deter-

mined in a sufficient amount to give pain and deafness, a paracentesis must be performed. This fluid, as a rule, is light and straw colored, seldom pus, but usually contains infecting organisms. The patient is often relieved after incision, but the fluid may change into pus within the course of forty-eight hours, and the ear needs careful watching. The change from serum into pus is frequently due to a reinfection of the middle ear from extraneous organisms in the external canal which, unfortunately, cannot be sterilized sufficiently to keep such infection from taking place. Many times there is a congestion of the mastoid cells with distinct tenderness particularly over the antrum and tip of the mastoid. This does not indicate the presence of an acute mastoiditis, but is merely an extension of the inflammation by contiguity. If a proper incision is made in the drum and the ear condition resolves, the inflammatory reaction in the mastoid cells resolves, too. Very often deafness persists for a considerable length of time, even after the discharge has ceased and all acute symptoms have disappeared. This is due mainly to a thickening of the drum and to adhesions of the ossicles consequent upon a serous effusion. Careful attention to this matter usually results in a complete subsidence of the symptoms.

When a purulent discharge takes place and the tenderness over the mastoid process increases in extent, we have an indication that some of the infection has found no outlet in the small honey-combed cells. If the discharge continues to be copious, if there is a great deal of pain with radiating headache, if there is tenderness on deep or superficial pressure which extends posteriorly behind the sinus, indications are present that a mastoiditis has developed which needs operation. However, many cases are seen with extreme tenderness over the mastoid where no operation is necessary. The writer recalls six cases of otitis media developing in one family, at the time of an epidemic of influenza. Three of the patients had marked symptoms of mastoiditis. One was operated upon. The other two refused operation and *got well*, although every indication was present that resolution would not take place without operative interference.

The X-ray picture of the mastoid is frequently of great help in borderline cases. One should not trust to an X-ray picture with-

out corroborative clinical evidence, of course, but it certainly makes assurance doubly sure. If a culture has been taken and the involving organism has been found to be other than the streptococcus capsulatus, one need not be alarmed; for the other bacteria, as a rule, do not give serious complications. When complications do occur, such complications demand immediate attention; for one may see develop a sinus thrombosis, a brain abscess, or acute meningitis—any one of which may result gravely.

PERIODIC PHYSICAL EXAMINATIONS

By J. MADISON TAYLOR

Associate Professor of Non-Pharmaceutic Therapeutics, Medical Department
of Temple University
Philadelphia

Occasional, or periodic examinations in one's later years are imperative; in early middle life essential; in early adulthood of the utmost importance.

They constitute the chief economic index of inherent resources; likewise they reveal where retrenchments must be made. No original capacities, no specialized faculties, no courageous self confidence, nor commendable efforts to keep well, can suffice to guard against the onset of insidious disease.

Nor is it a mark of valor to disregard powers of the enemy. We walk amid perils. We avoid many, 'tis true, but how pathetic is the episode of a robust, splendid citizen, falling suddenly by the wayside, involving disaster to many others; also important allied interests either suffer or are jeopardized by his collapse! The spectacle recurs constantly. Only rarely need it be.

Reasonable precautions will usually suffice to prevent such catastrophes. Especially is this true by foretelling and forestalling the approach of physical and also mental decrepitude.

Indeed, to become a derelict is almost worse than sudden death; which last, after all, may be regarded as a desirable form of exit.

Ramifications of correlated interests must always be considered. No one lives unto himself alone.

There are duties to one's household, one's company or one's clan which are as binding as duty to one's future state.

For the citizen, father, partner who values sustained efficiency, who wishes to learn how much of time or opportunity remains and would conserve these to the uttermost, there is one best means available, viz.: periodic careful evaluations of physical and mental assets.

By means of periodic examinations it can be determined whether one actually is the man he believes himself to be. If not, he should learn and trim his sails anew. Knowing precisely on what he may count, he may then revise methods to his lasting advantage.

If he has overstrained his physiologic credit, a change in course of living may readily lead to better things.

Definite retrogressions, even beginnings of what would inevitably result in decrepitude, can usually be delimited, or checked when taken in time. Nor is this candid seeking to learn what one hopes may not be, yet really is, in some form or degree, to be construed as timidity, or over-apprehension. On the contrary it is the mark of admirable judgment, and commendable prevision.

Biologic calculations have adduced evidence (however much concrete provings are needed) to the effect that man's possible years are near about 160 provided conduct could be made to accord with growth forces and survival values.

World thought runs now much toward eugenics; the science of inherent resources interpreted in the light of idealized environment and conformity in conduct to physiologic requirements. Among the fields of personal investigation easily the first are possibilities of dis-integrations in the cycle of structures immediately concerned in the maintenance of life, vegetative existence, breathing, digestion, circulation, elimination of waste matters and the like. This oftentimes constitutes rescue work, saving leaks heretofore undetected which may, and will, progress to irreparable losses.

Next in order of economic importance are the organs and structures of precision which should be conserved whereby proficiency is maintained, the even tenor of advance so desirable in one's career and pleasures. The eyes are usually reckoned the primal consideration; in some it may be the ear, in others the hand; in all the neuromuscular mechanisms.

We must not omit mention of the teeth, which are not only of exceeding use, but when seriously impaired, especially when wrongly assumed to be properly conserved, are not seldom causes of far

reaching disorder, of mental and physical catastrophes. One may reply that, nowadays, everyone is aware of the need for thorough investigation of the eyes.

Do not be misled. I have heard persons high in the scale of leadership boast that their eyes were so good as to need no help from medical experts; and for years they had avoided them. Let it be clearly understood that this is a grave blunder. There is no such critical index of aging, of loss of precious proficiencies, as advancing deterioration in the eyes. Since the great discovery of structural errors in the make-up of eyes, and how these may be corrected by the simple device of accurately fitted glasses, the age of individual usefulness has been almost doubled. Moreover, that terror which can make the boldest blanch, the onset of blindness in its various manifestations, causes and degrees, can only be determined by frankly seeking and submitting to expert tests. Take cataract for one illustration. The early use of suitable glasses alone may rescue an eye so threatened from destruction.

There follow many items of conservation (too long to present here) which bear upon both efficiency and survival. Habit formation is a great power for good and for evil. A heedless disposition, reckless tastes, pursuing lines of least resistance, haphazard adaptations, offer large fields for reaccounting and compulsory revision.

We need not take up here the obvious and much discussed questions of wrong habits of eating and drinking, of smoking and the like sybaritic trends. Less known, yet of equal significance are static errors due to domination of bad postural and motor habits.

Consider for a moment the body from the standpoint of a working machine devised to remain in commission, if rightly conserved, for at least three score years and ten.

This same body to do its perfect work must not only contain a set of sound organs whereby it shall perform serenely its appointed course, but there is also framework, thews, sinews, bones, a beautifully devised and perfected skeltal structure constituting an efficient container and ground work for these noble organs, and their biochemical complexities.

Man stands erect, the one and only animal which does. While he has evolved through untold ages, as the one erect, plantigrade mammal, whose component parts have adjusted themselves to ever

varying exigencies, reaching relative perfection, yet none the less certain penalties threaten the individual who omits to so revise conduct as to keep all this complex mechanism in order.

Man's upright posture while endowing him with definite kinds and degrees of preëminence places him also in some grave forms of disadvantage.*

This detriment is not so obvious if normal attitudes are maintained, but becomes forceful and hurtful when unnatural attitudes, amounting to deformities, are persisted in; and that too in spite of all the amazing endurance of occupational distortions. These induce anomalous compressions on tubular structures, angulations, kinks, adhesions and other positions of visceral disadvantage. Not seldom postural regulation alone is capable of restoring health.

An examination to be efficacious should include a careful estimation of the motor mechanisms beginning with the joints, flexures, tendons, their capabilities of movement, range, and the like. Closely associated is flexibility of the muscles, pliancy of structures concerned in the transmission and transformation of force. Many of the tendinous insertions will be found painful on pressure, effects of fibromyositis, producing limitations of movement which can and should be cured before permanent disabilities ensue. Capacity for movements in the thorax is highly significant. Impairments here make for obstructions to the excursus of the lungs, impediments to free action of the heart and great vessels, the very center of oxidation and oxygenation.

So also of the external abdominal muscles which should fully support the great vegetative and reproductive organs in order to render their action complete and harmonious. Undue relaxations here (unfortunately too common as middle age approaches) make for stagnation in the circulation, the splanchnic vessels in particular, inducing a form of neurasthenia most distressing and disabling.

Few remedial measures can accomplish more for general betterment than judicious training of these abdominal muscles, including also the diaphragm and lifting power of the thoracic and shoulder girdle group.

The erector spinae muscles need to be in good tone to maintain

*See paper by the author, "Orthotherapy, Normality of Posture as a Factor in Health," *Monthly Cyclo. and Med. Bull.*, Jan., 1914.

normal erectness, the uplift of the whole torso, sustaining thoracic competence and also interrelationships of the abdominal viscera. Hence it is obvious that scrutinization of the entire gross musculature is essential in determining degrees of organic competence. Elasticity is readily impaired as age creeps on. Undue compression on vital structures is hurtful in a thousand directions and advances insidiously interfering with ebb and flow of fluids, retarding elimination, holding back destructive waste products, encouraging cellular disintegration in organs whose action is essential to life itself. Obviously periodic examinations are gravely needed for every one entirely independent of any consciousness of disability.

Progress of Diagnosis and Prognosis

GENERAL METHODS OF EXAMINATION—SYSTEMIC AFFECTIONS—DISORDERS OF GENERAL METABOLISM

Comparative Determinations of Blood-Sugar by Polarization and Reduction—C. MAASE and H. TACHAU, *Zeitschr. f. klin. Medizin*, Vol. LXXXI, Nos. 1 and 2.

Comparative determinations of the blood-sugar by means of polarization and the reduction methods of Bertrand and Tachau gave identical results when the sugar content of the blood was normal or increased. In but one case the results were discrepant. In this case the sugar content determined polariscopically was decidedly greater than when determined by the reduction methods. This was probably due to the presence of a carbohydrate more markedly rotating, but weaker reducing than dextrose. After the ingestion of 100 grams levulose reduction showed decidedly higher values as polarization.

WESTERN.

Coagulation Factors in Hemophilic Blood—A. FONIO, *Mitteilungen a. d. Grenzgebieten d. Medizin u. Chirurgie*, Vol. XXVIII, No. 2.

The exact study of a case of hereditary hemophilia showed that the blood platelets were insufficient, but not decreased, that thrombogen was normal in its behavior, that thrombin was contained in the serum in large amounts, but that its activity was insufficient, and that fibrinogen was present in normal quantity. These factors tend to demonstrate that in hemophilia there is an insufficiency of the organs presiding over the organs generating the platelets. The characteristic phenomena of hemophilia may be explained on the hand of the insufficient function of the blood platelets.

MILL.

Leukocytes and Viscosity—A. GULBRING, *Beiträge z. Klinik d. Tuberkulose*, Vol. XXX, No. 1.

The viscosity of the blood is solely dependent upon the number of polynuclear leukocytes. The number of lymphocytes is without influence upon the viscosity. The greater the number of the polynuclear leukocytes the higher will be the viscosity of the blood.

FRY.

Colorimetric Determination of Urinary Uric Acid—H. F. Höst, *Zeitschr. f. klin. Medizin*, Vol. LXXXI, Nos. 1 and 2.

Author found the following modification of Riegler's method the most trustworthy of all the colorimetric tests for uric acid in the urine. Into test tube bearing the mark 20 cc. at the proper place, 2 cc. urine is placed. To this 0.6 gram ammonium chlorid is added. The mixture is then heated to about 40 deg. C., after which it is set aside for not less than half an hour. Then it is filtered through a small filter. A solution of ammonium sulphate (20 per cent.) for "Nachspülung" is then used 4 or 5 times. The ammonium urate on the filter is then brought into solution by means of boiling disodium phosphate (15 cc. of a 5 per cent. solution) which is filtered into the first test tube. A solution of phosphomolybdic acid (4 cc. of a 90 per cent. solution) is then added to the filtrate and the test tube filled to the mark 20 cc. with the disodium phosphate solution. The mixture is then heated to the boiling point, and when cooled off compared with the standard solution in the colorimeter. The standard fluid is composed of 2 cc. uric acid solution with 2 milligrams uric acid, 4 cc. phosphomolybdic acid and 14 cc. disodium phosphate solution.

WESTERN.

Grave Anemia in Childhood—E. STETTNER, *Jahrbuch f. Kinderheilkunde*, Vol. LXXX, No. 5.

Report of the hematologic examination of 3 cases of splenic anemia. Anemias in childhood often originate from increased disintegration of blood following infections. The prognosis is entirely dependent upon the degree of the affection. Therapeutic measures may be of great assistance.

MILL.

Phosphaturia—DÜNNER, *Berliner vereinigte ärztl. Gesellsch., Münchener med. Wochenschr.*, Feb. 2, 1915.

Author differentiates rigidly between the precipitation of ammonium phosphate (coffin-shaped crystals), the result of urea decomposition in bacteriuria, and genuine phosphaturia in which the phosphates are precipitated as salts of lime. When there exists a normal ratio of phosphoric acid and calcium the soluble double acid salts is formed. If there is an excess of calcium or a deficiency of phosphoric acid the insoluble simple acid salt is produced. Genuine phosphaturia is therefore divided into two groups: subacid phosphaturia (diminution of phosphoric acid) and calciuria. Genuine phosphaturia, a constitutional affection, must also be differentiated from alimentary phosphaturia. The decrease of acidity may be caused by acid impoverishment in the presence of increased hydrochloric acid excretion in the stomach. In the majority of the cases a direct influence of the nervous system upon the composition of the urine must be assumed. This explanation is

more plausible than the one which assumes a primary blood alkalescence which has as yet not been demonstrated. In calciuria the excretion of lime is augmented. The amount of CaO excreted in one day never exceeds 0.4 or 0.5 gram. The kidney excretes but 10 per cent. of the ingested lime. Increased ingestion of lime is not followed by an increased excretion of lime via the urinary system. Colitis does not stand at the foundation of phosphaturia and the kidneys do not exert vicarious function in respect to Ca excretion. Calciuria may exist without colitis. Phosphaturia is a partial manifestation of neurasthenia. The therapy must be directed against this affection. Phosphoric acid in 5 per cent. dilution, of which 20 drops is to be given three times a day, will be found of use in calciuria.

MILL.

Phosphaturia—ROTHMANN, Berliner vereinigte ärztl. Gesellsch., Münchener med. Wochenschr., Feb. 2, 1915.

There is no parallelism between neurasthenia and phosphaturia. Patients with phosphaturia, on the other hand, frequently show urticaria, eczema and vasomotoric disturbances.

MILL.

Phosphaturia—ROSIN, Berliner vereinigte ärztl. Gesellsch., Münchener med. Wochenschr., Feb. 2, 1915.

Phosphaturia is not a disturbance of metabolism, but a nervous excretory disturbance of the kidneys.

MILL.

Pseudo-Levulosuria—P. J. CAMMIDGE and H. A. H. HOWARD, Lancet, Feb. 17, 1915.

Although true levulosuria or fructosuria may be met with, it is apparently a rare condition and in the majority of cases, it seems probable that the term is a misnomer, and that the levorotatory reducing substance that occurs in the urine is in reality the ketonic acid, isoglucuronic acid, authors have described. This is differentiated from levulose by Borchardt's test, by being precipitated from an acid solution by saturation with lead acetate, and the melting point of the parabromphenylosazone.

SACHS.

Lipemia Retinalis—R. F. MOORE, Lancet, Feb. 20, 1915.

Lipemia retinalis occurs in cases of diabetes in young people who are usually bordering on coma. This condition is of grave prognostic significance. It implies a high grade of lipemia, such as probably only occurs in diabetes. The ophthalmic picture is so striking that it should not be mistaken for any other condition. The opacity of the plasma is most likely the cause of the conspicuous change in color and appearance of the retinal vessels.

SACHS.

Partial Myxedema—W. M. BARTON, Jour. A.M.A., Mar. 20, 1915.

The chief clinical types of thyroid insufficiency which appear to be recognized at present come under six categories: (1) Growing boys and girls, suffering either from general mental backwardness or persistent nocturnal enuresis; (2) Certain backward children between 2 and 5 years of age, showing various symptoms of which the two most familiar are slowness in learning to talk and delay in learning to walk; (3) Certain infants, who without being true cretins, present symptoms of amentia or idiocy; (5) Certain stout, sterile women of the child bearing age; (5) Certain females at or about the menopause who have rapidly become too stout; (6) Sufferers from certain nervous affections, which closely resemble and are frequently called neurasthenia and tic douloureux. SATHS.

Graves' Disease in an Infant Nine Months Old—O. KLAUS, Prager med. Wochenschr., Oct. 15, 1914.

Cases of Basedow disease are very rare in childhood. No case was ever described in a child 9 months old. The child exhibited the following symptoms of the disease: Pulse frequency 144, exophthalmus, distinct Gräfe and Stellwag's phenomena and enlargement of the thyroid gland. All these symptoms had vanished after one year. MILL.

Mutual Relations of some of the Glands with an Internal Secretion—L. OKINTSCHITZ, Archiv f. Gynäkologie, Vol. CII, No. 2.

It is as yet impossible to draw any conclusions concerning the antagonism or synergism of the glands with an internal secretion. Author's personal researches concerned the effect of the removal of the ovaries upon the thyroid, adrenals, hypophysis and uterus. Besides, observations were made as regards the influence of subcutaneous administration of the products of some of the glands (ovary, placenta, etc.) upon castrated animals. According to author, the corpus luteum is a gland with a negative internal function, i.e., a function neutralizing the toxic substances circulating in the organism. MILL.

Diagnosis of Carcinoma by the Abderhalden Method—S. CYTRONBERG, Mitteilungen a. d. Grenzgebieten d. Medizin u. Chirurgie, Vol. XXVIII, No. 2.

Report of 97 cases and detailed test protocols. Of 35 undoubted carcinoma cases 33 reacted positively. According to author the underlying principle of the method is absolutely correct, but the value of the reaction is limited on account of certain sources of error. MILL.

Faulty Vision and Ill-Health—W. W. KABAR, Med. Press (London), Feb. 10, 1915.

In endeavoring to relieve functional troubles, always keep in mind the etiological factor of the eye. Because a patient wears glasses does not necessarily mean that they are correct. As a matter of fact, not more than 25 per cent. of all glasses worn are in hailing distance of correctness. In case glasses do not relieve the symptoms, and you still have reason to suspect the eye, use the atropin test by instilling a one per cent. solution once a day for 10 days in both of the patient's eyes. This will in most cases paralyze the accommodation, stop the eye-strain, and in the latter case give immediate relief.

SACHS.

INFECTIOUS DISEASES

Relationship of Infantile and Adult Phthisis—J. P. CULLEN, Practitioner (London), April, 1915.

Adult and infantile phthisis are one and the same disease, the difference in the symptoms and signs are explicable on anatomical peculiarities. The affection in childhood may remain limited to the lymphatic paths. In many cases a diagnosis can only be made by means of radiography. Wrongly diagnosed or diagnosed too late, the parenchyma of the lung becomes affected. In childhood local symptoms are indefinite and general symptoms are more marked. In both adult and infantile tuberculosis, the path of infection is identical, in both it is at first pulmonary, and the point of entry is in the vicinity of the terminal bronchiole.

SACHS.

Tuberculosis of the Newborn—C. G. GRULEE and F. HARMS, Am. Jour. Dis. Child., April, 1915.

The newborn infant is affected clinically by the same infections in a much different way from the older infant. In the diagnosis of tuberculosis of the newborn, the combination of enlargement of the spleen, high irregular temperature and enlargement of the liver, together with tuberculosis in the mother, is suggestive. Little can be expected from the ordinary tests. In the newborn the von Pirquet test has proved negative almost without exception. A blood culture or the injection of blood into animals may be tried, but they offer many difficulties. Therefore it can be seen that it is very difficult to arrive at a diagnosis of tuberculosis in the newborn even though its presence be suspected.

SACHS.

Tuberculosis in the Aged—J. B. HAWES, 2nd, Am. Jour. Med. Sci., May, 1915.

The diagnosis of senile tuberculosis is often very difficult. Asthma, emphysema, and chronic bronchitis may so mask the tuber-

culous process in the lungs as to render its detection very hard. Constitutional symptoms may be lacking. Tuberculin tests are of no value. Röntgen ray examination is often of great service. Repeated and frequent sputum examinations are essential in these cases. Such examinations should be made at frequent intervals in every case of chronic lung trouble in elderly persons. SACHS.

Albumin Reaction of the Sputum in Pulmonary Tuberculosis—LÖWENBEIN, *Zeitschr. f. Tuberkulose*, Vol. XXIII, No. 2.

In the differential diagnosis between uncomplicated chronic bronchitis and tuberculosis an albumin content of 1 per mille and more positively points to tuberculosis. An amount of albumin below 1 per mille does not exclude tuberculosis. An albumin content of 1 per mille and more in chronic emphysema and bronchitis indicates that a tuberculosis is also present. Together with the other methods of examination the quantitative albumin determination furnishes valuable data as regards the prognosis. In general, the increase or decrease of the albumin content is of greater import than the absolute amount of the same. The alteration of the albumin content is often of greater import as is the increase or decrease of the tubercle bacilli. FRY.

Prognostic Value of Sputum Examinations in Pulmonary Tuberculosis—v. SZABÓKY, *Zeitschr. f. Tuberkulose*, Vol. XXIII, No. 4.

The albumin content of the sputum of tuberculous patients permits of prognostic conclusions. The larger the amount of albumin in the sputum the more unfavorable is the outlook in a case of tuberculosis. When the albumin content of the sputum is 1 per mille, or more, the course of the affection is always a more violent one. FRY.

Albuminuria in the Tuberculous—F. D'ONGHIA, *Beiträge z. Klinik d. Tuberkulose*, Vol. XXIX, No. 3.

In 50 young persons affected with tuberculosis albuminuria was found in 28 cases. FRY.

Tuberculosis and Gout—MAYER, *Zeitschr. f. Tuberkulose*, Vol. XXIII, No. 3.

In many cases there exists an interrelation between tuberculosis and gout. Examination of the purin metabolism will reveal interesting data. Hemorrhages as equivalents of gouty attacks may even supervene. FRY.

Studies in Pneumonia—R. COLE, *Johns Hopkins Hospital Bull.*, May, 1915.

It has so far been impossible to detect by chemical or biological means the presence of poisons, either in the media in which pneumococci have been cultivated or in the body fluids of animals

dying from infection with these organisms. Lately considerable stress has been laid on the importance of bacterial anaphylatoxins, as described by Friedberger, in producing the symptoms of infection. The anaphylatoxin theory assumes that the intoxication is due to split products of the bacterial protein. According to Friedberger the ferment causing the splitting is supposed to be present in the serum, but the demonstration by Neufeld and Dold, and also by Rosenow, that autolyzed bacteria, in the absence of serum, are toxic has required that the theory be modified so as to presuppose that the ferments are present in the bacterial bodies. Author has been able, however, to show that the bacteria simply dissolved in dilute solution of bile, or even brought into solution by freezing and grinding, are also toxic. Author concludes that the evidence is still very inconclusive, that any of the reactions is of significance so far as intoxication in lobar pneumonia is concerned. Experimental evidence indicates that the symptoms are due to the action of the living bacteria, rather than to the action of substances contained within the dead bacterial cells.

WESTERN.

Agglutination of the Spirochaeta Pallida—A. KISSMEYER, Deutsche med. Wochenschr., March 18, 1915.

Serum of syphilitics agglutinates the spirochaeta pallida in a specific manner. The reaction is not constant in syphilis, but may be demonstrated in all its stages. The relation of the agglutination to the treatment is not as yet determined.

MILL.

Positive Wassermann Reaction in Pemphigus—M. HESSE, Wiener klin. Wochenschr., Jan. 21, 1915.

Of 11 cases of pemphigus (vulgaris, vegetans, herpetiformis) 9 showed a positive Wassermann reaction. This reaction is, therefore, rather characteristic of pemphigus.

MILL.

Cerebrospinal Fever—E. HOBHOUSE, Brit. Med. Jour., March 6, 1915.

The diagnosis of cerebrospinal fever should not be influenced by the absence of any one or two supposedly essential symptoms; all are uncertain. But the combination of either rash, or vomiting, or squint, or rigidity of limbs, and opisthotonos, with a clinical picture otherwise resembling influenza, is very strongly suggestive of the graver disease.

SACHS.

Cerebrospinal Fever—M. FOSTER, Brit. Med. Jour., March 27, 1915.

Retraction of the head has been present in every case save one—that of a man found unconscious in bed. In 4 out of 19 cases, a definite rash appeared. It consisted of discrete papules about the size of number one shot, which did not disappear on pressure and varied in color from scarlet to mulberry. In the author's cases, there was only slight evidence of any affection of the cranial nerves.

The mental condition presented every gradation from profound coma with inability to swallow, through violent noisy delirium, to a mere condition of mental hebetude. Vomiting has always been present except in fulminating cases. Rigidity of the muscles of the limbs has not been a marked feature. The sphincters have been affected in a considerable proportion of the cases. SACHS.

Cerebrospinal Meningitis—A. LUNDI, D. J. THOMAS and S. FLEMING, Brit. Med. Jour., March 20, 1915.

The disease is much more widespread than is usually recognized. Authors say that the disease probably gives fair warning of its onset by catarrhal symptoms, and often goes no further. In its second stage it may run a long non-malignant course, giving plenty of time to arouse suspicion, and if correctly diagnosed would probably be cured by suitable treatment. During an epidemic, routine examination of all throats is very important. SACHS.

Streptococcus Meningitis—P. L. DU BOIS and J. B. NEAL, Arch. Pediat., Jan., 1915.

During the past four years authors have seen 18 cases of streptococcic meningitis, of which only one recovered. Five of the cases were due to the streptococcus mucosus capsulatus, the rest to the streptococcus pyogenes group. Of 11 cases, 5 gave a history of middle-ear disease or mastoid; 2 of scarlet fever, middle-ear disease and mastoid; one of operation on the nose; 2 of trauma of the head; one of whooping-cough. SACHS.

Schick Toxin Reaction—J. A. KOLMER and E. L. MOSHAG, Am. Jour. Dis. Child., March, 1915.

The reaction consists in the appearance, after the intracutaneous injection of a certain amount of diphtheria toxin, of an area of erythema with a brownish tinge, measuring 0.5 to 2 cm. in diameter, and accompanied by slight edematous infiltration of the underlying tissues. It appears in from 24 to 48 hours after the injection. This reaction is a reliable and valuable method for detecting susceptibility to diphtheria. Persons reacting negatively have an amount of anti-toxin in their system which is probably sufficient to protect them against infection. Persons reacting weakly or strongly positive may be regarded as susceptible to diphtheria. About 40 to 50 per cent. of children from one to 15 years old react positively to the toxin test; this means that the preliminary use of the toxin test will eliminate the necessity of administering prophylactic doses of anti-toxin to about 50 per cent. of the children. SACHS.

Diphtheria Bacilli in Herpetic Vesicles—RALL, Münchener med. Wochenschr., March 23, 1915.

Diphtheria bacilli are not rarely encountered in herpetic vesicles in

patients affected by diphtheria. No prognostic significance can be attached to this finding. Herpetic lesions carrying the diphtheria bacilli are probably never the transmitter of the infection. MILL.

Disturbances of the Internal Secretion in Dysentery—H. PRISER, *Deutsche med. Wochenschr.*, Jan. 21, 1915.

The disturbances of the internal secretion in dysentery observed by author concern especially the function of the thyroid-adrenal group. Disturbances of the pancreas-parathyroid group are much less in evidence. Many manifestations point to an increased tonus of the sympatheticus, others to an increased vagus irritation. These observations may also explain the good effects of preparations of belladonna in cases in which the opiates give no result whatever. Exophthalmos and a peculiar brightness of the eyes in the course or after recovery from dysentery seem to point to hyperthyrosis. The thyroid in these cases is frequently enlarged, but the thymus seems to be hypoplastic. A marked intolerance for carbohydrates is found in dysentery. MILL.

Latent Infection and Surgical Recovery—E. MELCHIOR, *Berliner klin. Wochenschr.*, Feb. 1, 1915.

The conception "recovery" is often quite an inadequate one. Generally speaking, a phlegmon, an osteomyelitis or a lymphadenitis is considered to be cured when the tangible clinical symptoms have disappeared. Such disappearance of the clinical manifestations, however, does not always correspond to a recovery in the bacteriologic sense. It may be but a latency, a semi-immunity, which may already contain the etiologic factor of a recurrence or of a metastasis. MILL.

RESPIRATORY AND CIRCULATORY ORGANS

The Auscultation Phenomenon over the Larynx in Croup and Pseudo-Croup—A. LEVINSON, *Münchener med. Wochenschr.*, Feb. 2, 1915.

The auscultation over the normal larynx of the child evinces in the inspiration period a dragging sound; when expiring the sound is somewhat lengthened. In the presence of pseudo-croup an impure, limited sound is heard during inspiration by auscultating directly over the thyroid cartilage. During expiration the sound is lengthened and dragging. Auscultating the suprasternal fossa moist medium râles are heard during expiration and inspiration, while over the lungs normal vesicular breathing is present. In the presence of croup the inspiration is an open vowel, deep and harsh. Expiration is much longer than inspiration and resembles a V. In case an advanced stenosis is present râles are not audible. Only when a bronchitis has supervened râles may be heard over the larynx, but the breathing always remains deep and dry. MILL.

Dyspnea and its Relation to Blood Reaction—T. LEWIS and J. BARCROFT, Quart. Jour. Med. (London), Jan., 1915.

Authors report 4 new cases in which the symptom-complex, previously described by them, was present. This complex is associated with reduced alkalinity of the blood and occurs in elderly subjects. It comprises a continuous dyspnea, often intensified for short periods, especially at night; good or bad blood aeration as judged by the absence of cyanosis, or such cyanosis as would seem compatible with simple cardiac dyspnea, and by examination of the alveolar air; Cheyne-Stokes breathing, with or without full apneic periods, and with an increase of pulse-rate (80-100) per minute; and lastly a subnormal temperature. This symptom-complex, associated as it is with cardiac enlargement, general arterial disease, and fibrosis of the kidney, may be, and frequently is combined with any of those symptoms spoken of at the present time as uremic. This association is not necessary, but is superadded to the symptom-complex. The complex is of extremely frequent occurrence. SACHS.

Differential Diagnosis Between Acute Abdominal and Acute Intrathoracic Disease—C. MACKENZIE, Lancet, April 17, 1915.

Severe abdominal pain, often accompanied by vomiting, rigidity of the muscles, and even local tenderness, may exist without any lesion of an abdominal viscus. It is advisable in every doubtful case to examine the thoracic viscera before submitting a patient to laparotomy. In all cases the pulse-respiratory quotient can be analyzed and considered with the temperature. If attention is paid to this quotient, many cases of pneumonia will not be treated by abdominal section. Reliance cannot be placed on the quotient or temperature where pleurisy or pericarditis are considered, and as in the past, so in the future will mistakes occasionally occur. A rectal examination should be made in all suspected acute abdominal conditions since tenderness is usually elicited, and actual swelling often found. SACHS.

Diagnosis of Mediastinitis—C. P. HOWARD, Johns Hopkins Hospital Bull., May, 1915.

The following conclusions are offered: Tuberculosis plays a very important part in the etiology of chronic mediastinitis. One or more of the pressure phenomena of mediastinal tumor and aortic aneurysm may be present in this condition. Perez' mediastinal friction may prove of diagnostic value. WESTERN.

Röntgen Ray and Pulmonary Conditions in Children—W. M. HARTSHORN, Am. Jour. Dis. Child., May, 1915.

The Röntgen ray is of distinct value in the diagnosis of diseases of the respiratory tract. In pneumonia a shadow may appear over

the suspected area several days before the development of definite physical signs. It is of value in determining the progress of lung involvement. Röntgenograms may be taken on successive days as long as there are signs of active advancement of the process. The röntgen ray offers material assistance in differential diagnosis, tuberculosis, abscess of the lung, lobar and bronchopneumonia. Through the obliteration of the costal-phrenic angle, the röntgen ray indicates the presence of an exudate. SACHS.

Capillary Pulsation—E. JÜRGENSEN, *Zeitschr. f. klin. Medizin*, Vol. LXXXI, Nos. 1 and 2.

Report of observations of a number of cases with capillary pulsation. Most cases were associated with arteriosclerosis. This has frequently ensued as a consequence of syphilis. The pronounced capillary pulse is a symptom of the much increased work of the left ventricle and the arterial circulation. Whenever the capillary pulse is met with, it points to disturbances necessitating a careful control of all the factors connected with blood movement and blood distribution. WESTERN.

Dilatation of the Arch of the Aorta in Chronic Nephritis with Hypertension—W. H. SMITH and A. R. KILGORE, *Am. Jour. Med. Sci.*, April, 1915.

Authors conclude that dilatation of the arch under the age of fifty years in non-syphilitic conditions is rather frequent, especially in chronic nephritis with hypertension. The dilatation may be quite marked, particularly in the younger patients. WESTERN.

Aneurism of the Sciatic Artery—R. C. BRYAN, *Annals Surg.*, Oct., 1914.

It has been noted that both spontaneous and traumatic aneurism of the sciatic artery occur more frequently in the male than in the female in proportion of two to one. Most of the patients are between 20 and 40 years of age in the laboring class and apparently otherwise healthy. It has also been established that aneurism of the sciatic artery occurs more often on the left side than on the right. D'Antona emphasizes the following: A line between the top of the great trochanter and posterior inferior spine of the ilium is a distinct landmark and separates gluteal aneurism which lies above, from sciatic aneurism which lies below this line. He also calls attention to the location of the bruit in reference to this line. D'Antona further states that a differential diagnosis between aneurism of the gluteal and sciatic artery is at all times difficult and in most instances impossible. Fischer says that aneurism of the gluteal lies high, as a rule, above the tuber ischii and to its inner margin, while that of the sciatic is deep, and at, or below, the level of the tuber ischii. Fischer also calls attention to the fact that with marked pulsation

of the tumor along the posterior border of the femur, increased thickness of the deep femoral and low pulse of the cruralis, one should bear in mind a possible ischiatico-popliteal aneurism. D'Antona lays particular emphasis on an early neuralgia, in cases of aneurism of the sciatic artery, on account of its normal anatomical relationship to the larger sciatic nerve. This obtained markedly in author's case and appears to be a strong diagnostic symptom for aneurism of the sciatic artery. He also refers to the hard edema which goes along with sciatic aneurism and which is produced by a vasomotor paralysis or vasodilating excitation which is not noted in the gluteal aneurism. Traumatic aneurisms, following stabs, gunshot wounds, or a fall, may be diffuse, circumscribed or varicose and are more rare than the spontaneous aneurism, whose history is negative to any injury but, in a certain percentage of cases, goes on to the formation of anastomotic aneurism. Fischer states that the differential diagnosis between aneurism of the gluteal artery, abscesses, cysts, and cancer, is always difficult and in some cases impossible. Diagnosis of aneurism of the sciatic artery must be based mainly on the complex of pulsations, bruits with aneurismal character, and sciatic pains which can be brought out in this region. Differential diagnosis should consider abscesses, hygroma, sciatic hernia, gumma, cancer (markschwämme) and pulsating sarcoma. Abscesses: In this region abscesses may be deep-seated, arising in the sciatic space by retroperitoneal involvement and extruded downward through the notch from above, or, more rarely, by way of the bowel. The rectovesical fascia, however, would appear to be a sufficient barrier against the escape of suppuration from the ischio-rectal space to this locality. The superficial phlegmons and cellulitis are to be recognized by local evidences with constitutional reaction. Rectal examination in both instances should be carried out. Bursæ: Various authors describe 31 bursæ about the hip-joint. Those which may be justifiably confounded with sciatic aneurism are: 1. The bursa of the gluteus maximus, which is located between this muscle tendon and the great trochanter; inflammation of this bursa is not uncommon, and gives rise to a doughy tumor behind the great trochanter with eversion and abduction of the thigh in contrast to the flexion and inversion of aneurism. 2. The bursa over the tuber ischii which is small, rarely involved, and points internal toward the anal margin along the lower fold of the buttocks. 3. The bursa of the gluteus medius developed in the tendon of that muscle as it runs over the upper and outer margin of the great trochanter. Inflammation here may be noted anterior and external to the trochanter. Sciatic hernia: Garré gives two forms of sciatic hernia, the hernia suprapyriformis and the hernia infrapyriformis. In both the intestine protrudes beneath the gluteus maximus and is deeply buried. Most sciatic herniæ occur in women past middle life. Beside the intermittent

pain which goes hand in hand with all varieties of hernia, in this instance there is a tumor below the gluteus maximus which is *reducible* with subsidence of the pain. Not infrequently these herniæ point inward to the anal margin and even to the coccyx. Gumma: Should always be suspected and a thorough investigation instituted. Malignant growths of any type may arise in this region, as nearly every form of tissue is represented here. Of the many varieties, pulsating sarcoma offers the greatest obstacle to differential diagnosis. In myelogenous sarcoma, pulsations and bruits are not infrequently present. True aneurism of bone, however, is extremely rare. Klebs reports no such case. Vibrating or pulsating sarcomata are due to great vascularity and cystic degeneration. The prognosis of aneurism of the sciatic artery is extremely profound. Spontaneous cure is not to be expected in any case. Very rarely does aneurism of this artery remain stationary. It may for a year or so, but fatal hemorrhage will result some time, and sudden rupture may cause death following a gradual asthenia. D'Antona has collected the figures of the mortality following ligation of the hypogastric artery and found it to be 40 per cent. The mortality of the cases in this series, surgical and medical, was 32 per cent. SACHS.

Contractility of the Heart—M. B. LEVITON, Jour. A.M.A., May 8, 1915.

The contractility of the heart muscle varies within wide limits, depending on the pathological process. This can be fairly well estimated clinically and is a valuable differential and prognostic sign. The findings on auscultation and percussion vary after exercise. The heart muscle may react in three ways: (a) In functional and early lesions, the heart boundaries may on exercise return immediately to normal, bruits disappear and normal tones at once replace them; (b) In moderately severe cardiac (and particularly cardiorenal) cases, the boundaries may return to normal not immediately but after rest in bed; (3) The heart remains permanently enlarged and inelastic, not only after the exercise test, but even on prolonged rest in bed. SACHS.

Paroxysmal Tachycardia—B. PARSONS-SMITH, Practitioner (London), April, 1915.

Too much weight must not be attached to the presence of murmurs in cardiac cases. A loud murmur may be produced by an insignificant lesion, and a soft murmur, heard only with difficulty, may possibly speak for gross and advanced disease. A heart, whose muscle suffers no loss of tone at the end of an ordinary day's work, is in a highly satisfactory state of compensation, whatever the lesion present. Our most valuable and reliable method of estimating cardiac tonus is by means of X-ray examinations, before and after exertion; for we know that a dilatation follows any appreciable loss

of tone, and that a heart whose tonic properties are up to the normal standard, not only fails to dilate, but may even diminish in size on physical exercise, assuming the latter to be short of actual strain. In any case of paroxysmal tachycardia, the following clinical varieties must be thought of: (1) Paroxysmal tachycardia of exophthalmic goitre; (2) associated with emotional states; (3) consequent on dyspepsia; (4) of auricular fibrillation; (5) of auricular flutter, and (6) dependent upon a transposition of the site of the origin of the heart's contraction.

SACHS.

Auricular Fibrillation—H. SCHOONMAKER, *Med. Rec.*, March 27, 1915.

Auricular fibrillation is the common cause of cardiac arrhythmia characterized by complete irregularity. Auricular fibrillation is of frequent occurrence, especially in mitral stenosis and in the senile heart. Auricular fibrillation should be recognized without the aid of recording instruments. An arrhythmia in which there is no regularity, no sequence, with the heart rate above 100, being uninfluenced by treatment, together with the positive or ventricular venous pulse, as seen in the neck is almost surely due to auricular fibrillation.

SACHS.

ALIMENTARY TRACT

Gastric Headaches—W. F. CHENEY, *Am. Jour. Med. Sci.*, May, 1915.

It is difficult to understand how disorder of the stomach can produce violent pain in the head, and no adequate scientific explanation for it can really be given; but it is equally difficult to explain in another case how constipation causes headache, and why the pain disappears promptly after the lower bowel is emptied. The headaches due to gastric disease are usually periodic. They repeat themselves throughout months or years. They may occur but once in a month, or once or several times in a week. Over certain periods they may become practically constant, though worse or better at certain times in the day. But the most common story is of sudden, unexpected attacks of pain in the head, coming after days or weeks of good health. Such headaches are of variable duration, but rarely last over 24 hours. The patient weakens with the pain in the morning, and it grows more intense as the day goes on; or it may come on in the evening, persist during the night, and pass off after the patient rises. These headaches may be so severe and prostrating as to incapacitate the sufferer for any kind of work while they last. Sometimes nausea and vomiting accompany the pain, giving rise to the popular term "sick headache." In other cases there may be no disturbance whatever of the stomach to make the digestive organs even suspected. Following the attack there may prevail a soreness over the scalp at the site of the previous pain and a feeling of

mental unclearness and confusion. The site of the pain is not sufficient to determine a gastric origin. Perhaps the most common type is hemicrania, one-sided pain, though not always of the same type. The pain is described as boring in character, through one eye or temple; or it may be the entire half of the head, even back to the base and the nape of the neck, that aches and throbs; while the opposite side is entirely free from discomfort and as clear as ever. But this one-sided headache is caused frequently by alimentary toxicosis; it is the characteristic type of the paroxysm known as migraine, and it sometimes occurs as a manifestation of uremia. Whether there is complaint of indigestion or not, a test meal and gastric analysis will be needed to reveal the cause of the headaches. The most frequent disturbance in such cases is one of motility; myasthenia or atony due to weakness of the muscular wall. The significant feature is the delay of food in the stomach. Hyperchlorhydria may be present in some of the cases. In the diagnosis of gastric headaches the history makes us only suspect, the gastric analysis makes us reasonably certain, but only after elimination of all other possible causes can this one be accepted as the basis of therapy.

WESTERN.

Familial Occurrence of Gastric Ulcer—PLITEK, *Archiv. f. Verdauungskrankheiten*, Vol. XX, Nos. 3 and 4.

Author diagnosed the presence of gastric ulcer in a number of members of one family. He opines that this occurrence is not an accidental one, as the entire family exhibited neuropathic phenomena. Besides local influences a pathogenetic influence of the nervous system must be accepted as of etiologic import; the familial occurrence of gastric ulcer is, therefore, not accidental.

WESTERN.

Familial Occurrence of Gastric Carcinoma—P. K. PEL, *Berliner klin. Wochenschr.*, March 22, 1915.

Of 7 children of healthy parents, 5 died of gastric carcinoma. Predisposing conditions could not be found. The remaining 2 children are still alive.

MILL.

Syphilis of the Stomach—M. EINHORN, *Med. Rec.*, March 13, 1915.

There is no absolute sign distinctive of gastric carcinoma (excepting microscopic tissue section) which may not be encountered in a gummatous tumor of the stomach. The absence of a positive Wassermann reaction does not positively militate against lues; nor does its presence indicate that the gastric affection is a syphilitic one. A vigorous antiluetic treatment carried out over an extended period of time (two or four weeks or more) gives us the desired information with regard to the differential diagnosis. If there is general improvement and with it a perceptible decrease or disappearance of the tumor, then we are entitled to make a diagnosis of gastric

syphilis. Patients with distinct syphilis frequently suffer from other organic lesions of the stomach which are entirely independent of the co-existing general lues. SACHS.

Tuberculous Stenosis of the Pylorus—W. PFANNER, *Mitteilungen a. d. Grenzgebieten d. Medizin u. Chirurgie*, Vol. XXVIII, No. 1.

Report of a case in which besides an apical affection there existed a stenosis of the first portion of the duodenum. The operation showed the presence of a tuberculous ulcer in the pylorus. This was removed, after which progressive improvement ensued.

MILL.

Fat Indigestion—C. H. DUNN, *Am. Jour. Dis. Child.*, March, 1915.

Fat tolerance is diagnosed in those cases in which the giving in the food of a moderate amount of fat is followed by the appearance in the stools of free fat or excessive soap, or in which an increase in the amount of fat given in the food produces immediately symptoms of indigestion and nutritional disturbance. There is no constant or typical appearance of the dejecta in these cases. Tuberculous babies are especially prone to fat intolerance. SACHS.

Influence of Posture on Indigestion in Infancy—C. H. SMITH and L. T. LE WALD, *Am. Jour. Dis. Child.*, April, 1915.

Röntgenography has shown the fallacy of the old idea that the stomach in infancy is vertical in position. The shape of the stomach depends on the amount of food and gas present, pressure from the outside, posture, etc. Authors conclude that air is swallowed with the food by many if not by all infants. The erect posture favors eructation of this air; the horizontal posture prevents it. The horizontal posture by preventing eructation is an important cause of vomiting, colic, indigestion and disturbed sleep. This is a very thorough and practical paper, and is profusely illustrated. SACHS.

Infantile Diarrhea—A. A. DAY and J. R. GERSTLY, *Am. Jour. Dis. Child.*, March, 1915.

Twenty-two cases of severe diarrhea studied by the authors divide themselves into three groups: (1) food disturbances; (2) infectious diarrheas, and (3) parenteral infections. By the latter is meant such cases of diarrheas as occur secondary to infections outside of the intestinal tract, as a bronchitis, an otitis media, or a choryza. But 2 of these 22 cases could be classified definitely as belonging to the group of food disturbances. These two showed the picture of alimentary intoxication described so clearly by Finkelstein—a rise in temperature, a sharp drop in weight, watery, green stools, perhaps even bloody, skin of a pale muddy color, fixed staring eyes, tireless deep breathing, an enlarged liver, leukocytosis and glyco-

suria. In two cases the gas bacillus was found. The greatest number of these cases were associated with foci of infection in other parts of the body, that is, the parenteral infection group. This corresponds with the observations of Finkelstein that the parenteral infections are by all means the most important factor in the production of food disturbances.

SACHS.

The Carmin Test and the Time for the Passage of Ingesta through the Alimentary Tract—H. STRAUSS, *Archiv f. Verdauungskrankheiten*, Vol. XX, Nos. 3 and 4.

It is true that the X-ray examination gives more complete evidence concerning the condition of the gastrointestinal canal than the carmin test. As regards the length of time a certain food remains in the alimentary tract, however, the carmin test which is so readily applicable gives just as reliable evidence as the X-ray examination. At any rate, the carmin test deserves a more frequent employment in the clinical study of the cases.

WESTERN.

Differential Diagnosis of Chronic Appendicitis—BISCHOFF, *Monatsschr. f. Geburtshilfe u. Gynäkologie*, Vol. XL, No. 3.

In 37 cases of uncertain diagnosis author employed the method of Bastedo. In 23 of these cases the method furnished positive results, and there was an appendiceal change in every one of them when operated upon. In 14 of the cases the method proved negative, and at operation no appendiceal manifestations were noted.

MILL.

Ulcerative Colitis—A. ALBU, *Mitteilungen a. d. Grenzgebieten d. Medizin u. Chirurgie*, Vol. XXVIII, No. 2.

A report of 23 cases observed by author. Of these 2 were acute, the rest chronic. One of the acute cases recovered; the other died. Of the chronic cases 6 recovered under exclusive medical treatment; 12, among which 3 cases were operated upon, did not recover, and 3 cases died. Ulcerative colitis is an independent, infectious disease of the intestine. Etiologically, it is probably the consequence of an infection due to the aliments. The ulcers, which are present in nearly every case, have the size of a pinhead, are superficial and occur in little groups. They occur most frequently in the region of the sigmoid flexure. Generally, they do not leave any visible scars. Clinically, the affection starts in most cases in a lingering manner, and manifests itself by tenesmus, watery and bloody, and slimy stools often containing pus and necrotic pieces of tissue. Constipation is frequently present. The X-ray will not reveal a typical disease picture.

MILL.

Intestinal Polyposis—W. C. CARRALL, Surg. Gynecol. and Obstet., April, 1915.

Intestinal polyposis is a comparatively rare disease. It occurs especially in the young and middle-aged. In the majority of cases the growths are found in the large intestine and in the rectum. The growths often cause obstruction and intussusception. The symptoms usually vary with the position, size and number of the polypi. Rectal polypi are usually easy to discover with a proctoscope. Regardless of the location of the growths, they usually produce hemorrhage sooner or later. Diarrhea may become very profuse at times, rectal tenesmus may be present, and vague abdominal symptoms, as colic and obstruction also occur. Of 52 cases reported by Doering, only one patient was perfectly well after four years.

SACHS.

Leukocytosis in Abdominal Hemorrhages—L. A. LEVISON, Jour. A.M.A., April 17, 1915.

Leukocytosis should not be relied on as a differential point when the clinical signs demand the differentiation of appendicitis or other inflammatory trouble in the abdomen and an intra-abdominal hemorrhage. Leukocytosis from the latter condition is to be distinguished from the post-hemorrhagic leukocytosis which follows any severe bleeding. Leukocytosis from intra-abdominal hemorrhage comes on within 24 hours and lasts until the second day.

SACHS.

Röntgen Diagnosis of Gall-Stones—L. G. COLE and A. W. GEORGE, Bost. Med. and Surg. Jour., March 4, 1915.

Experience has shown that gall-stones may be detected about twice as frequently as formerly by: (a) Special technic for making the röntgen plates; (b) careful study of the röntgen plates by various methods; (c) thorough intimacy with the röntgenographic appearance of gall-stones. A positive diagnosis can be made in so many cases that the negative diagnosis has become of considerable significance. If there is no direct evidence of gall-stones, the stomach, cap, duodenum, and colon should be examined for adhesions from an accompanying cholecystitis.

SACHS.

Tertiary Syphilis of the Liver—T. McCRAE, Johns Hopkins Hospital Bull., May, 1915.

The left lobe of the liver is often involved to a much greater extent relatively than the right lobe. This should always suggest the consideration of syphilis. A history of ascites which subsided spontaneously or disappeared after tapping to reappear sometime later should excite suspicion. The importance of examining the patient immediately after tapping also deserves emphasis. It may be possible only at that time to have an opportunity of palpating

the liver in a satisfactory way. After iodid is taken a remarkable feature is the rapidity with which the temperature falls to normal. In the majority of the cases this occurs within 2 days, and it rarely takes longer than 5 days.

WESTERN.

Pancreatic Disease—B. B. CROHN, Arch. Int. Med., April 15, 1915.

The quantitative examination of duodenal ferments is the most rational and accurate method of studying the external secretion of the pancreas. Diminution of such enzyme activity of the pancreas is a reliable sign of organic disease of the gland. Occasionally, though rarely, a diminution of ferments occurs as a symptom of organic disease elsewhere in the body. Roughly, the diminution of ferments is directly proportional to the extent of organic destruction which has taken place. The absorption of fat and nitrogen from the intestine is independent of the condition of the external secretion or even of its presence. Absorption may be poor with a healthy gland, or good with a gland of which only a fragment survives the disease. The functional activity of the gland determines the degree of absorption. Duodenal ferment tests give the index of the organic condition of the gland. Absorption tests give the index of the functional activity of the pancreas.

SACHS.

NERVOUS SYSTEM

Examination of the Reflexes—C. H. WÜRTZEN, Deutsche Zeitschr. f. Nervenheilkunde, Vol. LIII, Nos. 1 and 2.

Examination of 2,000 cases demonstrated the extraordinary constancy of the common skin and tendon reflexes. Patellar and plantar reflexes were always present, the tendon Achilles reflex was absent in 3.5 per mille. Abdominal reflexes could not be elicited in 1.61 per cent. and cremasteric reflexes in 2 per mille of the cases.

WESTERN.

Physiology of the Tendon Reflexes in Nurslings and Children—J. VAS, Jahrbuch f. Kinderheilkunde, Vol. LXXX, No. 4.

The tendon Achilles and patellar reflexes are the same in nurslings and children as in adults. In the few cases in which these reflexes cannot be obtained in healthy nurslings below six months of age, it is always the fault of external causes, as the technic of examination, the restlessness of the child, etc.

MILL.

The Oculo-Cardiac Reflex—E. B. GUNSON, Brit. Jour. Children's Dis., April, 1915.

The oculo-cardiac reflex is a reflex change in the rate of the heart associated in some cases with a change in rhythm, following ocular compression. The path of the reflex is considered to be

along the fifth cranial nerve, the medulla, and the vagus, or sympathetic. The reflex is positive when slowing of the pulse occurs and negative when either no slowing or actual quickening results. Persons exhibiting the former state are described as vagotonics, those exhibiting the latter as sympathicotonics. The reflex is positive on normal persons. The reflex is positive in about 92 per cent. of children convalescing from diphtheria and scarlet fever. In cases of so-called "cardiac paralysis" the reflex was negative and remained so until death. In cases which recovered, the reflex became positive when the heart returned to the normal state. SACHS.

Pharmacologic Tests in the Diagnosis of Disturbances in the Vegetative Nervous System—G. LEHMANN, *Zeitschr. f. klin. Medizin*, Vol. LXXXI, Nos. 1 and 2.

Examinations of 100 cases of manifold nature characterized by symptoms of disturbances in the vegetative nervous system. It was learned that the reaction to pilocarpin does not invariably correspond to the clinical diagnosis; frequently, the degree of this reaction does not go hand in hand with the clinical symptoms. Tests with atropin showed identical behavior. A positive atropin reaction by no means always corresponds to a positive pilocarpin reaction. There is no definite antagonism between the reaction to adrenalin on the one side and to pilocarpin and atropin on the other. Of 22 adrenalin-sensitive persons 21 showed a distinct pilocarpin reaction, while when there existed pilocarpin-sensitiveness a marked adrenalin reaction was hardly ever noted. The antagonism between vagotonia and sympathicotomy does not exist. Adolescents are more susceptible to pilocarpin and adrenalin; senile persons are susceptible to atropin. It is of therapeutic import that atropin is frequently of no avail in vagotonia, but that it improves the condition in certain instances, so that its administration should be tried. If improvement ensues the drug should be continued for a protracted period. Adrenalin, which is often useful in pronounced acute cardiac insufficiency, must be employed with caution on account of its frequent disagreeable secondary effects. WESTERN.

Paralysis Agitans—E. TRÖMMER, *Deutsche Zeitschr. f. Nervenheilkunde*, Vol. LIII, Nos. 1 and 2.

Author adds some as yet unobserved symptoms to the syndrome of paralysis agitans. He bases his observations upon a study of 40 pertaining cases. The new symptoms are: a preliminary stage often lasting for years and neurasthenoid in character, intention tremor, incongruence of tremor and rigor, certain trophic disturbances, abasia, Babinski phenomenon, epileptoid attacks, and peculiar psychoses in the course of the disease. WESTERN.

Paralysis of the Spinal Accessory Nerve—A. N. BRUCE, *Rev. Neurol. and Psych.*, Feb., 1915.

Paralysis of the spinal accessory from injury to the nerve during the removal of tuberculous glands from the neck is well known. The results, as a rule, become visible immediately after the operation. Author reports 2 cases in which the injury to the nerve was not evident until after 10 and 14 years respectively. The feeling of weakness in the shoulder joint has only been recently noticed.

SACHS.

Parenchymatous Syphilis—F. W. NOTT, *Brit. Med. Jour.*, Jan. 30, 1915.

It is rare to find an error in the diagnosis of general paralysis when in combination with the history and clinical signs and symptoms, the cerebrospinal fluid has been examined. Increase of globulin and phagocytosis do not of themselves prove the syphilitic character of a disease of the central nervous system. A positive Wassermann reaction in the cerebrospinal fluid was only found in cases of general paralysis, tabes, and syphilitic disease of the central nervous system.

SACHS.

Luetin Test in Late Syphilitic Disease of the Central Nervous System—V. KAFKA, *Berliner klin. Wochenschr.*, Jan. 4, 1915.

The luetin test, employed in 139 cases, gave positive reactions in 62 per cent. of latent general syphilis, in 72 per cent. of congenital syphilis, in 52 per cent. of general paralysis, in 90 per cent. of cerebral syphilis and 100 per cent. of tabes. The luetin reaction is hardly ever positive in the first or second stage of lues. On the other hand, in the tertiary stage it yields positive results in almost every case.

MILL.

Dyssynergia Cerebellaris Progressiva—J. R. HUNT, *Brain* (London), Vol. XXXVII, Part II.

There exists a chronic progressive form of cerebellar tremor, the most striking and characteristic symptom of which is a generalized volitional tremor, which begins locally and gradually progresses. In its advanced stage, the disorder of motility is comparable in severity and violence with that of Huntington's chorea, or the generalized athetosis. There is, however, this difference, that in a position of rest and muscular relaxation, the tremor movements cease. An analysis of the motor disorder shows a marked disturbance of the ability properly to control and regulate coördinated movements. This is shown by the presence of hypermetria, dysmetria, adiadosis, dyssynergia, hypotonia, and intermittent asthenia. All of these symptoms, including the volitional tremor, coincide with the classical symptomatology which results from a loss of the cerebellar control over voluntary movements. The disorder is therefore re-

garded as of cerebellar origin. The local onset, gradual progression, and chronic course indicate a progressive degeneration of certain special structures of the cerebellar mechanism presiding over the control and regulation of muscle movements. SACHS.

Spinal Cord Tumors—J. COLLINS and H. E. MARKS, *Am. Jour. Med. Sci.*, Jan., 1915.

Pain and other classical data are valuable, but not essential phenomena in the early diagnosis of spinal cord tumors. The essential element in the diagnosis is the determination of a gradually progressive motor and sensory spinal paralysis, the upper pole of which, despite increase in cross-section intensity, varies slightly, if at all. In every case of so-called transverse myelitis, the possibility of cord tumor should be considered. The diagnosis of myelitis of unknown origin is made far too often; exploratory laminectomy is not done often enough. Painless advancing tumors are not atypical. SACHS.

Night Terrors in Children—T. A. WILLIAMS, *Med. Press* (London), April 14, 1915.

Author relates a number of cases of night terrors in children which illustrate the great sociological importance of conditions purely psychological. Impaired efficiency from psychological causes is quite common in children. When a nervousness of this origin shows itself, a proper analysis of the child's mind is the first step towards its removal. SACHS.

URINARY ORGANS—MALE GENITALIA

Renal Functional Tests—J. T. GERAGHTY, *Johns Hopkins Hospital Bull.*, May, 1915.

Functional studies reveal only the excretory capacity of the kidney. By themselves they do not make the diagnosis or settle the prognosis. The value of any of these excretory tests is purely empirical, because of lack of sound physiological information dealing with the ultimate physics and chemistry of the excretion of any substance by any part of the kidney, the tubes or glomeruli. They indicate only the functional value of the kidney at the time at which the test is performed, but cannot of themselves indicate what the renal function will be to-morrow or next week. This latter information is to be derived from the knowledge of the underlying pathological process which is producing the reduced function. WESTERN.

Syphilitic Nephritis—A. STENGEL and J. H. AUSTIN, *Am. Jour. Med. Sci.*, Jan., 1915.

There is evidence to suggest that there exists a parenchymatous type of nephritis due to syphilis, characterized by an abundant albu-

minuria, with many hyaline, granular, and occasionally epithelial casts, with a tendency to produce edema of renal distribution, associated, as a rule, with moderate reduction of phthalein output and exhibiting an almost constant tendency to the presence of doubly refractile lipoid globules, varying in size from an erythrocyte, to globules three or four times this diameter. These globules sometimes float free in the urine, but many times are a constituent of a compound granular cell or possibly of an epithelial cast. On the other hand, similar lipoid globules may be found in severe acute or chronic parenchymatous nephritis of other etiology, but in only a minority of the cases.

SACHS.

Tubercle Bacilli in Urine—L. BROWN, Jour. A.M.A., Mar. 13, 1915.

No staining method differentiates absolutely tubercle bacilli from smegma bacilli, but cultural methods may aid greatly. Animal inoculations with the production of tuberculosis is an absolute test, but of value only when positive. The same care about the collection of urine should be exercised as about the collection of sputum. Tubercle bacilli can be excreted through apparently normal kidneys. Radiography may aid in the quick detection of caseous foci when the urine contains no tubercle bacilli. Tubercle bacilli occur in the urine in genital tuberculosis usually late in the disease and are consequently of little aid in the diagnosis of the condition.

SACHS.

Renal Tuberculosis—G. S. GORDON, Surg. Gynecol. and Obstet., Feb., 1915.

Renal tuberculosis gives no local symptoms in a large percentage of the cases. Hematuria or symptoms of its spread to the bladder may be the first manifestations of its presence. Gonococci, staphylococci, or other pathologic microorganisms in the urine in no way exclude the concurrent presence of tubercle bacilli. Author thinks that renal tuberculosis predisposes the urinary tract to other infections.

SACHS.

Pyelitis—C. POSNER, Berliner klin. Wochenschr., Jan. 18, 1915.

Author is not willing to determine whether pyelitis is of cystic or hematogenous or lymphogenous origin. Both eventualities are possible, but the metastatic development appears to be the most frequent.

MILL.

A New Preparation for Pyelography—E. N. YOUNG, Bost. Med. and Surg. Jour., April 15, 1915.

Collargol is an absorbable kidney poison when used in the renal pelvis. The emulsion of argentide as prepared by Mr. Godsoe, the pharmacist at the Massachusetts General Hospital, is a clear non-

absorbable opaque fluid, which can be used with the minimum of danger to the patient and maximum satisfaction to the surgeon. Silver iodid itself cannot be used for this preparation, but an accurately made saturated solution will do very well. Since the latter is somewhat difficult to make, and if not correctly made, may be irritating, author has simplified the formula by using a saturated solution already on the market. Godsoe prepares the emulsion as follows: Quince seed 100 grains, water 8 ounces, macerate for 24 hours with frequent agitation. Do not crush the seed. Strain through cloth. Add 2 per cent. boric acid solution up to 20 ounces. It is important to extract with water and not with boric acid solution, as boric acid solution does not make a good mucilage. Enough of this mucilage is added to 12½ c.c. of argentide to make 50 c.c. and vigorously shaken for two minutes. The resulting emulsion lasts several weeks and is a thin clear fluid, flowing freely through a ureter catheter.

SACHS.

Diverticulum of the Bladder—H. CABOT, Bost. Med. and Surg. Jour., March 11, 1915.

Author reports ten cases of diverticulum of the bladder. The average age of onset of the symptoms was 37 years. Author is of the opinion that these sacculations are of congenital origin. They may and do exist for years without causing symptoms which, when they appear, depend upon the advent of infection. They occur most commonly in the neighborhood of the ureteral orifices, and may sooner or later by pressure upon the ureter produce ureteral dilatation, hydronephrosis, and extensive destruction of the kidney. Those cases which have been discovered early and removed have been followed by complete cure.

SACHS.

Testicular Tuberculosis—J. S. McARDLE, Practitioner (London), April, 1915.

The external secretion of the testicle is not the only one which renders the gland of importance to the well-being of the subject, for while in the lower animals emasculation has few, if any, ill effects, in man it produces a profound mental depression, at times of an alarming nature. Mental weakness and even mania have followed in the track of castration. The affections which most stimulate tuberculosis of the testicle are syphilis of that organ and gonorrheal epididymitis. The differential diagnosis is rarely difficult, for in tuberculosis the epididymis is the primary center, in syphilis the testicle proper is the point of deposition of the infective material. In tuberculosis, the testicular substance is usually encrusted upon only by pressure.

SACHS.

FEMALE ORGANS OF GENERATION—PREGNANCY—
PARTURITION—INFANTS

X-Ray Diagnosis in Gynecology—I. C. RUBIN, Surg. Gynecol. and Obstet., April, 1915.

Author employs the X-ray in gynecological diagnosis in conjunction with intra-uterine collargol injections. A 10 per cent. collargol solution is essential for a satisfactory X-ray picture. Under mild pressure (i.e., about 3 m.m. of mercury) the injection is not attended by pain. It is not desirable to inject more than 5 c.c. of the solution in the average case. The cases should be selected. One should make sure that there is no active infection of the uterus or the tubes. Post-abortive conditions with fever are contraindications. There are no bad sequels, the menstrual cycle is not disturbed, and the method is safe as far as peritonitis is concerned. The method is of aid (a) in the diagnosis of the patency or the occlusion of the tubes; (b) in differentiating intra-uterine from extra-uterine tumors as intra-ligamentous cysts and myoma; (c) in certain malformations of the uterus and possibly also of the tubes; (d) in determining whether a single or bilateral salpingectomy has been done on a patient that had previously been operated; (e) in studying true flexions of the uterus and mal-developments.

SACHS.

Diagnosis and Prognosis of Renal Changes in Pregnancy—WOLF and ZADE, Monatsschr. f. Geburtshilfe u. Gynäkologie, Vol. XL, No. 5.

The various forms of renal disturbances occurring during pregnancy cannot be clearly differentiated at the present day. A chronic nephritis may develop from a nephritis during the puerperium. Albuminuric retinitis may also be observed in uncomplicated nephropathy and eclampsia. In chronic nephritis and synchronous pregnancy the occurrence of albuminuric retinitis has not the bad prognostic significance as in the absence of pregnancy. It may entirely disappear after labor.

MILL.

The Total Cholesterin of the Blood in Obstetrical and Gynecological Cases—M. HUFFMANN, Zentralblatt f. Gynäkologie, 1915, Nos. 2 and 3.

The cholesterin content of the blood increases during pregnancy by about 0.06 per cent., reaches its maximum during the last month and declines to the normal amount after 8 to 10 days from the time of delivery. In eclampsia especially high values appear to occur. The cholesterin curve is apparently not influenced by menstruation. It is markedly increased in narcoses and declines in the presence of malignant growths, especially when there is synchronous anemia or cachexia.

MILL.

Recent Publications

New Books

DISEASES OF THE BRONCHI, LUNGS, AND PLEURA. By FREDERICK T. LORD, M.D., Visiting Physician, Massachusetts General Hospital; Visiting Physician, Channing Home (for Consumptives); Instructor in Clinical Medicine, Harvard Medical School. Illustrated with 93 Engravings and 3 Colored Plates. Philadelphia and New York, Lea and Febiger, 1915.

A man possessed of the vast clinical experience of Dr. Lord should have overcome his natural timidity and presented to us in the very first instance the results of his own work and observation. Instead of doing this, he has chosen to publish a very diligent retrospect on the diseases of the respiratory organs which is based almost exclusively upon the literary production of others. It is really a pity that the author kept himself so much in the background. However, it must be frankly stated that his work is the only complete and up-to-date book on the subject which has recently been published in the United States.

H. S.

INFANT-FEEDING. Its Principles and Practice. By F. L. WACHENHEIM, M.D., Attending Pediatricist, Sydenham Hospital and Mount Sinai Dispensary, New York City. Philadelphia and New York, Lea and Febiger, 1915.

This is an unusually lucid recapitulation of nearly all that has been published on the question of infant-feeding during the past ten years. Although the author advances no theory of his own—and this is hardly necessary in view of the fact that we can do very well without a dozen or two that have been solely advanced for the promulgation of the greater glory of the respective originators—he has succeeded in presenting the subject matter in a novel, and at the same time, concise manner. The little book cannot fail to make numerous friends among those who are brought in frequent contact with healthy and diseased babies.

H. S.

DIFFERENTIAL DIAGNOSIS. Volume II. Presented through an Analysis of 317 Cases. By RICHARD C. CABOT, M.D., Assistant Professor of Clinical Medicine, Harvard University Medical School, Boston; Chief of the West Medical Service at the Massachusetts General Hospital. Profusely Illustrated. Philadelphia and London, W. B. Saunders Company, 1915.

Dr. Cabot continues his scheme to teach what he considers "Dif-

ferential Diagnosis" by means of the analysis of case histories. The method, originated by the author, in order to be of real benefit to the reader, demands extraordinary memory on the latter's part. Besides, it is rather too elementary and does not convey any mode of procedure in the diagnosis of cases with which the modern clinician had not become acquainted in his college days. H. S.

THE BACKWARD BABY. A Treatise on Idiocy and the Allied Mental Deficiencies in Infancy and Early Childhood. Awarded the Alvarenga Prize of the College of Physicians of Philadelphia. By HERMAN B. SHEFFIELD, M.D., Author of *Modern Diagnosis and Treatment of Diseases of Children*, etc. New York, Rebman Company.

This well written and illustrated prize-essay is a careful study of mental deficiencies in infants in contradistinction from that in older children. Stress is put on environment as a predominating cause of mental backwardness, and the author maintains that, while the Darwinian theory of heredity holds good for normal racial characteristics, it is not applicable to abnormal mental and physical states. In the early detection of mental deficiencies in infancy the author suggests a number of valuable mental tests, and describes a peculiar attitude—*status idioticus*—assumed by these children which is pathognomonic of their condition. Under the heading of "moramentia" the diverse forms of delayed mentality from sense deprivation, etc., are fully gone into, and a new "incentive" method of training is outlined which is based upon using food as a bait, as it were, to induce mental defectives to learn to help themselves, and the like. Special attention is given to organotherapy and surgery in the management of idiocy and cognate mental degeneracies. H. S.

DIE ERKENNUNG UND VERHÜETUNG DES FLECKTYPHUS UND RUECKFALLFIEBERS. Von Generaloberarzt Prof. Dr. L. BRAUER, Eppendorf. Beratender innerer Kliniker bei der Armeearbeitung Woyrsch. Nebst Vorschriften zur BEKAEMPfung DER LAEUSEPLAGE BEI DER TRUPPE, Von K. u. K. Regimentsarzt Dr. JULIUS MOLDOVAN, Präses der Salubritätskommission der 2. österr.-ungar. Armee, Mit 4 farbigen, 1 schwarzen und 1 Kurventafel sowie 5 Abbildungen im Text. Würzburg, Verlag von Curt Kabitzsch, 1915.

Of all medical publications that were prompted by the present war this brochure on typhus fever is without doubt the most interesting one which has reached the reviewer's table.

In Germany and Austria typhus exanthematicus is of very rare occurrence. In times of peace sporadic cases of the disease may be encountered in the main centers of traffic in the Russo-Polish frontier districts. The infection, on the other hand, is endemic in the Balkan countries, in Southern Russia and in Poland, including Warsaw.

Typhus fever is said to hardly ever occur in the Summer; it is, however, quite frequent in the latter part of the Winter and in the Spring. Under the influence of unsanitary conditions extensive epidemics may then suddenly arise.

The causative agent of spotted fever is as yet unknown. The Wassermann reaction is generally negative in this disease. Salvarsan is therapeutically ineffective. For this reason it is unlikely that any of the species of spirochetes is etiologically responsible for the affection.

The virus is probably attached to the leukocytes and not to the red blood cells. The free blood serum and the spinal fluid are apparently not infectious.

The transmission of typhus fever may occur in two ways, viz., by the bite of *pediculus vestimenti*, the clothes louse, and that of *pediculus capitis*, the common or head louse. Lice are true transmitting agents; they become active infective agents only five or six days after they have taken up the infected blood. The virus is still demonstrable in the second generation of the lice. It has not been positively shown that bed-bugs or fleas may transmit the disease.

Lice do not inhabitate straw for any length of time. They also never deposit their eggs in the straw. It is a mistake to believe that the infected straw on which soldiers have been resting is one of the main sources of the spread of the lice. The contagion takes place from man to man or through the agency of infected woolen underwear.

H. S.

THE PRINCIPLES AND PRACTICE OF TOOTH EXTRACTION AND
LOCAL ANESTHESIA OF THE MAXILLÆ. By WILLIAM J. LEDERER,
D.D.S., Dental Consultant to the German Hospital in the City of New York.

This little volume of 258 pages will prove not only of interest to dentists, but really ought to find its way into the library of the medical man. It is a short but full description of all types of tooth extractions, ranging from simple cases to those types proving surgical operations. The author describes each class of cases, giving his technic of procedure. The second half of the book treats

on local anesthesia, especially "nerve blocking" as applied to the jaws, a feature which should interest every surgeon, as this type of anesthesia replaces narcosis in almost all jaw operations. The volume is rich in exceptionally good illustrations.

S. E. F.

MEDICAL ETHNOLOGY. By CHAS. E. WOODRUFF, A.M., M.D., Author of "The Effects of Tropical Light on White Men" and "Expansion of Races;" Associate Editor, "American Medicine"; Lieutenant-Colonel, U. S. Army, Retired; Member American Therapeutic Association, etc. New York, Rebman Company, 1915.

The author makes the following introductory remarks: "Medical ethnology deals with the different morbidity and mortality rates of the different physical types of people living in the same locality. Demography treats of the changes in a population as a whole, its increases and decreases through births, deaths and migrations. Medical ethnology is then a branch of demography, and explains why certain changes of type take place. These changes have been known for a very long time, indeed ever since ethnography has been a science, but it is only recently that their causes have been discovered and the matter found to be of great therapeutic and hygienic importance."

It is, indeed, a great undertaking for a physician to tackle a problem that can only be mastered by the most advanced and profound of biologists, and while Dr. Woodruff's attempt is certainly a very courageous and creditable one, it must necessarily lack the great guiding principle, the common denominator which a physician, educated in the era of medical casuistry, will hardly ever find in the domains that are not particularly his own.

The book, however, imparts a great deal of interesting and valuable information not only to the practitioner of medicine, but also to the lawyer, the theologian and the educated public in general.

H. S.

New Editions

DISEASES OF THE HEART. By JAMES MACKENZIE, M.D., F.R.C.P., LL.D., etc., Physician to the London Hospital (in Charge of the Cardiac Department); Consulting Physician to the Victoria Hospital, Burnley. Third Edition; Second Impression. London, Oxford Medical Publications, Henry Frowde, Hodder and Stoughton, 1914.

The first edition of this work marked a distinct era in the proper appreciation and the rational treatment of the diseases of the heart.

Mackenzie is indeed the founder of modern clinical cardiology. The present edition records all the advances pertaining to the subject that have been made since the past four or five years. Progress in clinical cardiology has chiefly taken place in three directions. First, as far as a succinct differentiation of disease-signs is concerned; second, as to the relation of heart symptoms to eventual heart failure, and, third, as to the scientific foundation of cardiac therapy. The present edition abounds in common sense, and though the subject in its modern aspects is naturally a complicated one, especially for the older men in medicine, the masterly fashion in which it is treated and the classical, simple language employed by the author will overcome the intrinsic difficulties which interfere with a ready understanding, and render the perusal of this important work a veritable pleasure. H. S.

URINARY ANALYSIS AND DIAGNOSIS BY MICROSCOPICAL AND CHEMICAL EXAMINATION. By LOUIS HEITZMANN, M.D., New York. Third Revised and Enlarged Edition. With 131 Illustrations, Mostly Original. New York, William Wood and Company, 1915.

Instead of the third, the present edition of "Heitzmann" should be the fifteenth. The comparatively slow sale of this, the most important work on urinary microscopy in existence, demonstrates clearly that the average practitioner of medicine has not as yet awaked to the necessity of a more refined examination of the urine. This, I am sure, does not pertain to the readers of the ARCHIVES OF DIAGNOSIS who know Dr. Heitzmann's theories and interpretations from his articles and illustrations that have appeared in its columns. There can be no doubt that the future belongs to the urinary microscopy as taught by Heitzmann. H. S.

DIAGNOSTIC AND THERAPEUTIC TECHNIC. A Manual of Practical Procedures Employed in Diagnosis and Treatment. By ALBERT S. MORROW, A.B., M.D., Clinical Professor of Surgery in the New York Polyclinic; Attending Surgeon to the Workhouse Hospital, and to the Central and Neurological Hospitals. With 860 Illustrations, Mostly Original. Second Edition, Thoroughly Revised. Philadelphia and London, W. B. Saunders Company, 1915.

On the advent of the first edition of this book we were glad to be able to recommend it to our readers (ARCHIVES OF DIAGNOSIS, 1911, p. 212). The present edition contains about 60 more pages and 45 additional illustrations. The author has given undue prominence

to some of the devices of his medical friends and associates, while he has omitted to give mention to certain diagnostic and therapeutic technic procedures that should have been included in a book of such broad scope.

H. S.

LEHRBUCH DER SPEZIFISCHEN DIAGNOSTIK UND THERAPIE DER TUBERKULOSE. Für Aerzte und Studierende. Von Dr. BANDELIER, Chefarzt des Sanatoriums Schwarzwaldheim in Schömburg bei Wildbad und Prof. Dr. Roepke, Chefarzt der Eisenbahnheilstätte Stadtwald in Melungen bei Kassel. Achte Auflage. Mit einem Vorwort von. Wirkl. Geh. Rat Prof. Dr. R. Koch, Exzellenz. Mit 25 Temperaturkurven auf 7 Lithographischen Tafeln, 2 Farb. Lith. Tafeln und 6 Textabbildungen. Würzburg, Verlag von Curt Kabitzsch, 1915.

The fact that this work on specific diagnosis and treatment of tuberculosis has appeared in its eighth edition is in itself sufficient proof of its value and popularity. It certainly needs no recommendation on our part.

Among the last sentences of the book are the following: Tuberculin must become an integral part of the armamentarium of *every* physician, must become the alpha and omega of our diagnostics, prophylaxis and therapeutics of tuberculosis. Only then it will accomplish that for which it was designed, viz., to assist in the extermination of tuberculosis."

H. S.

INFECTION AND IMMUNITY. A Text-Book of Immunology and Serology. For Students and Practitioners. By CHARLES E. SIMON, B.A., M.D., Professor of Clinical Pathology and Experimental Medicine at the College of Physicians and Surgeons; Pathologist to the Union Protestant Infirmary and the Hospital for the Women of Maryland; Clinical Pathologist to the Mercy Hospital of Baltimore, Maryland. Third Edition, Revised and Enlarged. Illustrated. Philadelphia and New York. Lea and Febiger, 1915.

When we reviewed the second edition of this work we said that the author has clarified a complex subject in an admirable manner. This is also the case in the present edition, which, in some respects, even surpasses the former. The section on the Wassermann reaction, for instance, has been almost entirely rewritten. The theory and technic of Abderhalden's protective (defensive) ferments have been dwelled upon in sufficient detail, and other recently advanced factors pertaining to infection and immunity have found due consideration.

The subject-matter, as set forth, is readily grasped by any practitioner who is not entirely fossilized.

We congratulate Dr. Simon not only upon his excellent work, but also upon his moral courage in "respectfully dedicating" it at this time to the atrocious, heinous Teutons "Paul Ehrlich, the Grandmaster of Experimental Medicine and the German Men of Medical Science."

H. S.

A TEXT-BOOK OF THE PRACTICE OF MEDICINE. For Students and Practitioners. By HOBART AMORY HARE, B.Sc., M.D., Professor of Therapeutics, Materia Medica, and Diagnosis in the Jefferson Medical College of Philadelphia; Physician to the Jefferson Medical College Hospital; One Time Clinical Professor of Diseases of Children in the University of Pennsylvania, etc. Third Edition, Revised and Enlarged. Illustrated with 142 Engravings and 16 Plates in Colors and Monochrome. Philadelphia and New York, Lea and Febiger, 1915.

It is always a pleasure to consult one of Dr. Hare's books. His style is classical in its simplicity, and I doubt very much that there is another medical author in this country whose writings can compare with those of the Philadelphia clinician as far as succinctness, straightforwardness and lucidness are concerned.

The third edition of this Practice of Medicine is a beautiful volume which is entirely brought up to date. It serves the purpose for which it is intended very well.

H. S.

THE ARCHIVES OF DIAGNOSIS

A QUARTERLY JOURNAL DEVOTED TO THE STUDY
AND THE PROGRESS OF DIAGNOSIS AND PROGNOSIS

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FOUNDED AND EDITED BY
HEINRICH STERN, M.D., LL.D.
New York



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Special Articles

STUDIES IN THE SCHICK DIPHTHERIA REACTION

By ABRAHAM LEVINSON

AND

MORRIS L. BLATT

Chicago

Von Pirquet's discovery of the cutaneous tuberculin reaction, in 1907, paved the way for the investigation of specific skin reactions in infectious diseases. Since that time we have had many contributions in this direction. Noguchi ⁽¹⁾ described his luetin test for the detection of syphilis; Irons ⁽²⁾ obtained a cutaneous reaction with a glycerin extract of gonococcus in persons suffering from gonorrhea; Claypole ⁽³⁾ obtained a reaction in persons affected with a streptothrix group of organisms.

One of the most important skin reactions described recently is one obtained in the absence of sufficient diphtheria antitoxin in the blood. B. Schick, the discoverer of the test, found ⁽⁴⁾ that diphtheria toxin in minimal lethal doses, when injected intracutaneously, will produce a specific reaction which can in turn be influenced by the injection of diphtheria antitoxin. We are already in possession of many data regarding this test. There still remains, however, quite a fruitful field for investigation. We undertook a series of tests in our hospital and dispensary cases, with the following problems in mind:

1. What percentage of individuals possess natural immunity, the ages at which immunity is most common, and what is the effect of sex on natural immunity.

We were especially interested in noting the applicability of the test in dispensary practice where patients are not under constant observation as they are in the hospital, the result of this test in turn to be of service in indicating its usefulness in private practice.

2. Whether a disease other than diphtheria has any suppressing effect on the Schick reaction.

3. Whether a v. Pirquet test done simultaneously with the Schick has any influence on the Schick reaction.

4. Whether tuberculin treatment has a counteracting effect on the Schick.

5. What effect vaccination has on the Schick.

6. How different members of the same family react to the Schick.

Before presenting our findings and observations, we shall give a brief résumé of the test in general.

PRINCIPLE

It has been a matter of common observation among physicians that some individuals do not contract diphtheria even though exposed to it. Römer (⁵) has described a biologic test for the determination of the amount of antitoxin in the blood. He found that intracutaneous injections of toxin and of human serum into a guinea pig resulted in necrosis if the serum did not contain sufficient antitoxin, but was neutralized if it did. Schick found that the intracutaneous test when applied to human beings gave the same information as to the presence or absence of antitoxin in the blood.

In making the test on a guinea pig that weighed 250 grams, Schick used 1/50 of a lethal dose of toxin. For instance, if the lethal dose for the guinea pig was 0.005, 0.1 c.c. of a toxin 1/1,000 dilution was used. Some patients to whom the test was applied reacted positively, others negatively. In testing the blood of those who showed a negative reaction to this toxin injection, by the Römer method, Schick and numerous other observers found that the blood contained at least 0.03 unit of antibodies or antitoxin pro cmm. A

positive reaction, on the contrary, showed that the blood contained less than 0.03 unit of antitoxin pro cmm., the amount required to produce an immunity to the toxin injection. It was also found that a larger dose of antitoxin will suppress the toxin reaction much sooner than a smaller dose, and that some individuals require less antitoxin than others to bring about this result. From a series of experiments along this line, Schick concluded that the principle of antitoxin reaction in man is the same as that in the guinea pig, that first the toxin in the tissues is paralyzed and then the toxin in the blood.

TECHNIC

The technic of different workers on the Schick reaction varies slightly, especially in the amount of diluted toxin administered. Park (⁶), for instance, dilutes the toxin so that 1/50 of the lethal dose is contained in 0.2 c.c. of the solution. Since we, however, adopted the method used by Schick, we shall give a description of his technic.

A strong diphtheria toxin is taken, and the minimal lethal dose for a guinea pig weighing 250 grams is determined by injecting a series of guinea pigs. The dilutions are then made with sterile normal salt solution until 0.1 c.c. of the solution equals 1/50 of the minimal lethal dose. The toxin should be kept in a cool place, to prevent spoiling. It is also best to keep the mixture in a dark bottle, as light seems to have a deleterious effect upon it. A rubber cap through which the needle may be inserted before using is preferable to a cork, as with each opening of the bottle there is danger of contamination.

A syringe divided into 1/10 c.c. graduations can be used. More accurate results, however, may be obtained from the use of the tuberculin syringe, which is graduated into 100 parts. The needle is of great importance in this work. It should be very thin and have a short point, so that when introduced into the skin the point may easily be covered over by the skin, thus preventing the fluid from running out around the point. The injection with a needle of this description, besides causing less pain than the injection with a large needle, also greatly decreases the possibility of trauma, a matter that usually presents difficulty in ascertaining the reactions.

The best place for injection is in the scapular or intrascapular region. The needle is inserted intracutaneously, the skin about the region of injection being held between the thumb and the index finger. If the injection has been made correctly, the skin about the area of injection will show a whitened appearance with definite indentations. Some workers use the arm as a place of injection. Our experience, however, makes us favor the back as the region of injection, for the reason that the skin there being less sensitive the danger of traumatic reaction is much less than it would be in the arm or forearm.

Reaction, as a rule, occurs in about twenty-four hours after injection, although some reactions may occur as early as twelve hours and others as late as seventy-two hours after injection. A positive reaction is manifested by an area of erythema and induration, which usually becomes more marked the following day and persists for five or six days. The reaction seen in twenty-four hours presents an erythema of from 0.5 cm. to 1 cm. long, and from 0.2 to 0.8 cm. wide, with some induration. The twenty-four hours following, the erythema shows an increase in strength and diameter. The third twenty-four hours, the erythema takes on a brownish tinge, becoming darker with each succeeding day until its disappearance, leaving a brownish discoloration at the place of reaction, or resulting in a superficial cracking of the skin over the spot of injection with a subsequent desquamation in eight or nine days.

The extent of the reaction is of course a matter of great importance to the worker. It involves the determination of the reaction as to its positive or negative character, often a puzzling question to answer. A reaction that clearly shows the findings described above can easily be characterized as positive, just as one that shows merely the point of injection can be put down as negative. There are cases, however, that show a slight reaction, i.e., a faint erythema, and in these the question arises as to whether the reaction is positive or negative. Schick has used as control on the opposite side a toxin-antitoxin mixture, consisting of a solution of toxin with an excess of antitoxin, to make sure that the positive reaction, on the other side, if it does occur, is due to the toxin. We, however, found that besides the difficulty in getting toxin-antitoxin, the result may be misleading, as the antitoxin

may, and, in fact, does at times give a serum reaction. We, therefore, prefer as a control on the other side a normal salt injection, which we have found quite satisfactory. Moreover, the control can usually be omitted, the toxin test alone being sufficient.

We took the following as our standard of characterization:

Positive Reaction

Erythema and induration, at least 0.5×0.2 cm.

Negative Reaction

Needle track, redness or induration.

Small papule at point of injection, showing no increase subsequently.

Erythema without induration, disappearing without leaving trace in 24 hours.

FINDINGS

We diluted a strong diphtheria toxin with 19 parts of normal salt (1 to 20) and this solution we diluted further with 28.5 parts of normal salt solution; 0.1 c.c. of this solution was, therefore, equivalent to 1/5,900 c.c. of the toxin ($1 \times 20 \times 28.5 \times 10$). All other steps in our technic corresponded to the technic of Schick described above.

We tested 208 children. Of this number 60 were normal, 55 were afflicted with diseases other than diphtheria or tuberculosis, 86 were tuberculous or suspects on whom a v. Pirquet was done, 6 had been vaccinated a few days before the Schick test was applied, and one was a case of diphtheritic paralysis.

Series I consisting of 60 normal children between the ages of 7 months and 9 years gave 47 or 78.13 per cent. negative and 13 or 21 $\frac{2}{3}$ per cent. positive.

Sex played no rôle whatever in the result of the reaction. Age, on the other hand, did. All the children under one year of age showed a negative reaction, the susceptibility increasing with advance of age.

In series II out of 55 children afflicted with diseases other than diphtheria and tuberculosis, 34, or 61.8 per cent., showed a negative reaction to the Schick test.

Here also, as in the case of normal children, sex exerted no

influence. Age was a factor, all children under 6 months reading negatively.

The disease seemingly plays no rôle in increasing the susceptibility of the Schick reaction. The higher percentage of positives in this series (38 per cent., as compared to 21 per cent. in normal cases) may be accounted for by the greater number of older children examined.

In series III 33 children, suspects of tuberculosis, received the Schick simultaneous with the v. Pirquet test. They seemed to have no influence upon each other, some children giving a positive Schick and a negative v. Pirquet, and vice versa.

In series IV out of 38 children that received tuberculin treatment, 16 were positive. The result, however, bore no relation to the reaction obtained from the tuberculin treatment.

CONCLUSIONS

1. The Schick reaction is valuable in dispensary, as well as in private practice.

2. One should wait at least twenty-four hours before deciding whether a Schick reaction is positive or negative. The examination should be repeated the following twenty-four hours, and if possible also several days later. This, of course, diminishes somewhat the value of the Schick test as a diagnostic measure in urgent cases of diphtheria.

3. While a positive Schick reaction does not necessarily mean that the disease in question is diphtheria, antitoxin should be given in all cases showing a positive Schick reaction upon exposure to diphtheria.

4. Our series of cases showed a percentage of negative Schick reactions ranging from 61 per cent. to 78 per cent. This would seem to indicate that a very high percentage of children possess natural immunity to diphtheria. The administration of antitoxin can, therefore, be eliminated in a great many cases if the Schick test is applied.

5. The Schick reaction, as a rule, is negative in children under six months of age, frequently also in children under twelve months. Susceptibility, however, increases with increase of age.

6. Sex exerts very little influence on the Schick test.

7. Diseases other than diphtheria have no effect on the Schick reaction.

8. Tuberculin tests, as well as tuberculin treatment, exert no influence on the Schick test.

We wish to acknowledge our indebtedness to Dr. George Weaver of the Durand Hospital, who supplied us with the diphtheria toxin needed for our work.

We also wish to express our thanks to the medical staff and nurses of the Sarah Morris (Michael Reese) Hospital, the Jewish Aid Dispensary and the Bethlehem Nursery, for the assistance rendered us in making our tests.

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(Editor's Note).—The tables accompanying this article were omitted for technical reasons.

THE DIAGNOSIS OF ABNORMALITIES OF MYOCARDIAL FUNCTION

By T. STUART HART

Assistant Professor of Clinical Medicine, College of Physicians and Surgeons,
Columbia University; Visiting Physician, Presbyterian Hospital
New York

V. PAROXYSMAL TACHYCARDIA

Acceleration of the heart rate, which has been discussed in the last paper, is exceedingly common and is important as a symptom associated with many conditions. Paroxysmal tachycardia, which we are now to examine, is relatively rare, and is associated with

phenomena so distinct and definite that the syndrome deserves consideration as a clinical entity.

This group is particularly characterized by the suddenness of the change in the rate of the heart. The acceleration in rate occurs as a paroxysm whose onset is abrupt and whose termination is equally sudden. The change in rate, both of the onset and offset of the attack, occurs in a period of time less than that occupied by one normal cardiac cycle. The duration of the paroxysms are extremely variable. They may last for only a few beats or may continue for minutes, hours or days. The longest attack which has come under my notice was continued for 28 days. This variability is the rule not only comparing different cases, but also in the successive attacks of a single individual. The relative time consumed by the paroxysms and the intervals of slow rate is very variable, but in nearly all instances the slow periods exceed the paroxysmal periods by a considerable margin.

MECHANISM

An analysis of the paroxysms shows that it is composed of a series of contractions having their origin in some part of the cardiac musculature other than the sinus node; in other words, a rapid succession of extra systoles; in some point of the heart wall excitability is raised to such a point that for a period stimuli are set free at an abnormally rapid rate, and, in accordance with the law that the most excitable portion of the heart sets the rate for the less excitable portions, this excessively irritable point usurps the function of the pace-maker, and for the time the normal pace-maker, the sinus node, is buried in the flood of stimuli arising from this new point of origin. Usually all of the contractions of a given paroxysm arise from a single point and spread over the heart muscle by the same path. This is shown by the similarity of the waves obtained in graphic records. For the most part, the contractions are rhythmic, hence their rate is to a degree a measure of the rate of stimulus formation and the excitability of the irritable point.

During the period of slowing, the sinus node regains its ascendancy and sets the pace. If one studies carefully the periods of slow rate, one will almost invariably discover isolated extra sys-

toles occurring more or less frequently. These are usually of the same type as those which go to make up the beats of the paroxysm, and are often of material assistance in determining the particular point in the heart in which the extra systoles of the paroxysms have their origin. It is conceivable that any portion of the heart muscle may be capable, under suitable conditions, of assuming the rôle of pace-maker for a limited period of time. We are certainly able to define paroxysms which have their origin in the wall of the auricle, in the region of the auriculo-ventricular node and in the right and left ventricles. Most of the paroxysms have an auricular origin. Ventricular paroxysmal tachycardias are comparatively rare.

When the point of origin is in the auricle, the ventricle usually responds promptly and in the usual manner to each auricular impulse. At times, however, the electrocardiographic records suggest that the stimulus has taken a path through the ventricle wall, somewhat removed from the normal, or again the exciting effects of the frequent stimuli may be seen in a depression of the bundle contractility, as evidenced by an abnormally long period between the auricular and ventricular contractions.

It has been shown by Erlanger¹ that stimuli may pass over the conducting system of the heart in a direction opposite to the normal. We have evidence that this occurs in paroxysms of ventricular origin, and that the auricular contraction is a response to stimuli reaching it from the ventricle.

EXPERIMENTAL PRODUCTION

In a previous paper it has been pointed out that single extra systoles may be produced experimentally by applying mechanical or electrical stimuli to various portions of the cardiac musculature. If a properly spaced series of such stimuli are applied to the wall of the heart, a tachycardia will instantly result, composed of a succession of extra systoles. During such an artificial paroxysm, the activity of the normal pace-maker is submerged by the stimuli set free from the new focus. When the artificial stimuli are withdrawn the tachycardia terminates abruptly. The normal pace-maker immediately regains its ascendancy and the normal rhythm

¹Arch. Int. Med., 1913, Vol. XI, p. 362.

is resumed. Such paroxysms may be induced by stimulation of either the auricle or the ventricle. When the ventricle is thus excited, the stimuli are transmitted upward to the auricle, a direction the reverse of the normal, and the contractions follow instead of precede the ventricular contractions. These retrograde stimuli pass the bundle of His with less velocity than those which pass over the heart in the normal direction, hence a part of them may be blocked and the auricle may fail to respond to each ventricular contraction. Tachycardias have been experimentally produced by the administration of aconitin (Cushny), muscarine (Rothberger and Winterberg), by an abrupt increase of the blood pressure (Hering), and by ligation of the coronary arteries (Lewis); a production of attacks of tachycardia by ligation of the coronaries particularly elicits our interest, since it more nearly approximates conditions which we may encounter clinically. Lewis² found that obstruction of the blood flow in the right coronary was usually, and that of the descending branch of the left coronary was invariably, followed by isolated ventricular extra systoles, as the nutrition of that portion of the ventricular wall supplied by these vessels became progressively impaired, extra systoles appeared at shorter and shorter intervals, until finally there was established a rapid series of rhythmically recurring extra systoles, constituting a true paroxysmal tachycardia. Under these conditions the stimuli became retrograde and the auricular followed the ventricular contraction. The extra systoles were rhythmical and graphic records showed that in a given case all the extra systoles had a single point of origin. In dogs rates between 300 and 420 per minute were obtained. The phenomenon occurred both when the vagi were intact and when they were sectioned, showing that the disturbance had its origin in the wall of the heart and could not be ascribed to altered central innervation. When the ligature was removed and the circulation became re-established, the paroxysm abruptly ceased and the sinus node resumed its function of pace-maker.

The diagrams, figures 1 and 2 indicate the mechanism of the paroxysmal attacks. Figure 1 represents a focus of abnormal irritability situated in the wall of the auricle. The impulses are set

²Heart, 1909-10, Vol. I, p. 98.

free so rapidly that the stimulus material forming at the sinus node is destroyed before reaching maturity. As soon, however, as the abnormal irritability of the auricular wall is lost, the accumulation of stimulus material at the sinus node continues for the normal period and thus the node resumes its rôle of pace-maker. Figure

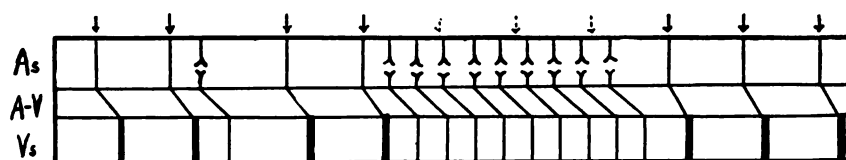


FIG. 1

Diagram showing mechanism of auricular tachycardia. One isolated extrasystole is indicated and a short paroxysm composed of a rhythmic series of similar extrasystoles. The temporary pacemaker is located in the wall of the auricle. The conduction time ($A-V$ period) is lengthened during the paroxysm.

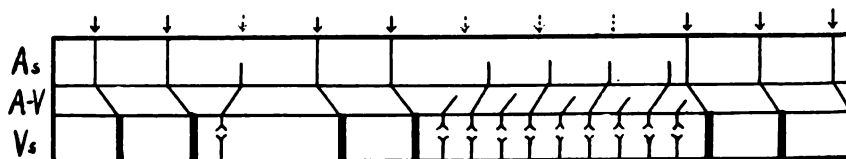


FIG. 2

Diagram showing the mechanism of ventricular tachycardia. One isolated ventricular extrasystole and a short paroxysm composed of a rhythmic series of similar extrasystoles. The temporary pacemaker is located in the ventricular wall. During the paroxysm the auricle contracts in response to "retrograde stimuli" passing upward from the ventricle, every other impulse from the ventricle is blocked.

The arrows indicate the points of origin and the direction taken by the stimuli. Dotted arrows indicate the time at which the normal stimulus at the sinus node should reach maturity if its formation were not interrupted by the extrasystole. The thickness of the lines representing ventricular systole indicate the relative effect of the normal beat and the beats of the paroxysm in maintaining an adequate circulation. As = auricular systole. $A-V$ = time of transition from auricle to ventricle or the reverse. Vs = ventricular systole.

2 represents an abnormal focus in the ventricular wall, which, for a short period becomes the pace-maker of the whole heart. Here the ventricular impulses become retrograde, that is, they passed upward over the $A-V$ bundle and stimulated the auricle from below. These impulses are frequently blocked, as is indicated in the diagram, in which the auricle responds only to every other ventricular impulse.

It might be supposed from this review of the mechanism of these disturbances that paroxysmal tachycardias would be frequent sequelæ of single extra systoles. This is not the case. Isolated extra

systoles are extremely common. Probably most individuals reaching the age of 50 have had extra systoles at one time or another, but attacks of true tachycardia are comparatively rare. On the other hand, it may be said that probably every true paroxysm is preceded by isolated extra systoles.

PATHOLOGY

Little is known of the histological changes which may form the anatomical basis of paroxysmal tachycardia. In my own series only two cases have had a fatal termination, and in neither of these was a post mortem permitted. In the literature several autopsies have been reported and these have all shown more or less excessive myocardial change—sclerosis, fibrosis, atrophy, and arterial degeneration, particularly of the coronaries. One does not feel that we have as yet evidence of any definite pathological lesion which is characteristic. Experimental evidence suggests that the cause may be found in the intracellular chemical change induced by variations in the blood supply in the heart, which may or may not show degeneration of the myocardium.

ETIOLOGY

In no one of my series of 26 cases of paroxysmal tachycardia have I been able to obtain a history of a similar condition in an ancestor or in any immediate relative. My youngest case was a boy who had his first attack when 10 years of age; the oldest a man of 69, whose paroxysms had annoyed him for 2 years. One patient, a man of 44, has suffered from attacks over a period of 20 years. The distribution by decades of the time of onset in my series is as follows:

Decade	10-20	20-30	30-40	40-50	50-60	60-70
Number of Cases....	4	7	4	4	5	2

Among the 26 cases which I have observed, 19 were males and 7 females. The following tabulation indicates that the syndrome occurs about twice as often in men as in women.

	Hoffmann ¹	Lewis ²	Hart	Total
Men	6	18	19	43
Women	4	11	7	22
				—
				65

An analysis of my cases presents the following factors, which may have a bearing direct or indirect on the condition of the myocardium. Alcohol was used to excess by 4; tobacco by 2 of the men. Severe gastrointestinal disturbance had preceded the attacks for several years in 3 of the women; nearly all had a history of one or more of the infectious diseases of childhood; in one case the onset of tachycardia followed 6 months after a severe infection of the middle ear; in another yellow fever antedated the attacks by 2 years. There had been frank attacks of acute articular rheumatism, followed by endocarditis with valvular defects, in 4 cases; a syphilitic infection was demonstrable in 4 cases, 3 of which showed evidence of myocardial damage other than the attacks of tachycardia. Several of the series had taken considerable doses of digitalis; in one a physician whose arrhythmia had been wrongly diagnosed as complete irregularity and auricular fibrillation had taken very large doses, and it seems to me that this was undoubtedly an important agent in increasing the irritability of the heart muscles. The attacks in the youngest patient of my series, a boy of ten, immediately followed a race in which he participated, at which time the physician who saw him found evidence of acute dilatation. A case of mild Graves' disease, in which the pulse averaged 100, has shown on several occasions paroxysms lasting only a few minutes in which the rate was between 160 and 170. Valvular defects were present in 9 of my patients; the mitral valve was involved in 7, of which 4 were cases of well-marked stenosis; one patient had an aortic insufficiency and one had defects of both the aortic and mitral valves; 15 cases showed various degrees of cardiac enlargement. In many cases the irritability of the heart muscle seems to require a very small exciting factor to induce an attack. The patient will usually ascribe the onset to flatulence, some emotional disturbance or unusual

¹Die Electrocardiographie, Wiesbaden, 1914.

²Clinical Disorders of the Heart Beat, London, 1913.

physical exertion; any one of these is probably an efficient cause to call forth an attack in a myocardium suitably damaged.

SYMPTOMS

The symptoms associated with paroxysmal tachycardia are of great variety, and show great differences from individual to individual. This is doubtless in a large measure due to the extent of damage present in the myocardium and the ability of the heart to meet the tax thus exacted. The patient is practically always conscious of the abrupt onset and termination of the attacks. They usually describe the attacks as beginning with one or two "thumps" or "throbs" in the precordial region, followed by a sensation of fluttering in the chest, which is terminated by another "thump" or "flop," and the attack is over. The amount of anxiety is always greater in the early attacks; as the patient becomes more or less accustomed to the paroxysm he is less alarmed, and a momentary pause in his activities may be the only evidence to show that he knows the attack is on. This absence of alarm I have noticed particularly in young adults who have had attacks for a number of years, but whose hearts show no anatomical abnormality and functional disturbance characterized only by the attacks of extra systoles at more or less infrequent intervals.

One of my patients, whose attacks have continued for several days, was quite unconcerned even when his heart was beating at 170. He rarely voluntarily assumed a recumbent position on account of the attacks and it was difficult to convince him that rest at these times was important.

In those who have an associated valvular lesion, and in those with evidence of marked arterial changes, a greater discomfort and attendant anxiety are closely associated with the symptoms referable to the cardiac insufficiency which is induced by, or the precordial pain which accompanies, the attack.

At the onset patients often complain of palpitation in the chest and a swelling and pulsation of the vessels of the neck. Often they have eructations of gas, nausea and vomiting. There may be a "gone," sinking feeling, and, if the attack is prolonged, sweating, coldness, great lassitude and an intolerable feeling of weakness. They may have a sensation of palpitation or of bounding in

the chest, shortness of breath or a sensation of suffocation. In one case under my observation attacks were invariably accompanied by a watery diarrhoea; in another by frequent micturition.

In the prolonged attacks, increase of the cardiac dulness to the left can sometimes be made out and the symptoms of circulatory embarrassment terminate the picture. The veins are not properly emptied, but are engorged, and there is pronounced cyanosis. The liver may be increased in size and become tender to palpation. There may be edema of the extremities; there may be cough with profuse thin, or blood-streaked, expectoration, with the physical signs of pulmonary congestion.

The paroxysms are often attended with headache and dizziness, more rarely with momentary or prolonged periods of unconsciousness, which may be explained on the basis of cerebral anemia. Pain is sometimes prominent. Sometimes this is accompanied by the feeling of oppression and of constriction of the chest, which accompanies the attacks, in nearly all patients to a greater or less degree. The pain is usually precordial, and is sometimes sharp, suggesting a real angina, and may radiate into the arms and back and sometimes one can detect areas of hyperesthesia over the chest and arms, following the distribution of one or more of the upper thoracic and lower cervical nerves. Some patients complain of numbness and tingling of the extremities.

A progressive cardiac insufficiency may terminate in general anasarca, pulmonary edema, collapse and occasional death. As a rule, however, the signs of cardiac insufficiency are very moderate, and even when present to an extreme degree clear up with great rapidity, following the abrupt ending of the rapid heart action. The absence of alarm, the facial change of expression from one of anxiety to complete calm; the abrupt change from dyspnea to quiet breathing; the sudden cessation of pain; the subsidence of engorged veins of the neck coincident with the termination of the paroxysm present some of the most remarkable and agreeable clinical phenomena with which we are familiar.

The signs of pulmonary congestion and edema of the extremities may require a period of days for their subsidence, the rapidity depending to a considerable degree on the functional efficiency of the heart when it has resumed its normal rate.

As illustrating the character of severe attacks terminating fatally, one case which I had the opportunity to observe closely for a period of months, may be described.

A man, 55 years of age, who had a leutic infection 20 years earlier, had a heart moderately enlarged to the left and a faint systolic murmur at the apex. Between the attacks his pulse was about 70 with many extra systoles. At all times there was evidence of a moderate degree of cardiac insufficiency. A description of the attacks, obtained from the patient, was as follows:

"The exact cause of these attacks of syncope and tachycardia, which come as often as twenty times in one day and have been absent as long as 26 days, cannot be determined. Many times he has been awakened from his sleep by dizziness to become unconscious and have a typical attack. Again, a slight exertion, as walking, going up stairs or straining to pass water, may be followed by an attack, but these same exertions, or even more severe ones at another time, may have no harmful effect. The attack comes on suddenly with dizziness, grayness before the eyes and a buzzing in the head like an organ. There are no premonitory symptoms. Unconsciousness follows rapidly, and when he comes to his heart is beating very rapidly, 200 to 250 to the minute. There is a choking sensation, as if a ball were in the throat, and he is shaking all over. There is never any pain over the heart or down the arm. At times he has been struck down as if by electricity without warning, again he has simply had dizziness and grayness, without losing consciousness. The tachycardia lasts a varying length of time, sometimes for only ten minutes, at other times all day. During its continuance he has great gastric disturbance, with frequent vomiting. He cannot forecast the end of the attacks until it is at an end. Then, at times, a violent regular beating of the heart is succeeded by two or three irregular beats, as if something shook the heart, and this is immediately followed by two or three tremendous throbs of the heart with each of which there is a feeling as if fresh air were forced into his throat and head and the attack stops suddenly as it began."

His paroxysms of tachycardia continued for 5 years, becoming gradually more frequent, and he finally died during an attack.

IDENTIFICATION

The conditions other than paroxysmal tachycardia which afford a heart rate of over 160 are extremely rare. During the paroxysm the pulse is exceedingly small, often irregular in force and frequently cannot be detected at the wrist. Under these conditions our examination should at once be directed to the precordial region. The apex beat may be imperceptible to the touch or, when palpable, may give the impression of complete irregularity. The heart sounds may be indistinct and have a fetal character; often they are sharp and distinct; as a rule, they are perfectly rhythmic, but so rapid that the rate can be only approximately estimated; this is best accomplished by counting short (5 seconds) periods. If one is fortunate enough to be making observations at the beginning or at the termination of the attack, the change in rate is readily detected. The transitions are usually accompanied by one or two large forcible beats, with loud sounds and unusually large pulse waves. The change in rate is quite abrupt. In the absence of such an observation the patient will frequently establish the diagnosis by his description of the sudden onset and termination of the attacks. Valvular murmurs, if present during the slow rate, sometimes cannot be detected during the paroxysm. In some cases a heart without murmurs during the slow period will develop a loud systolic murmur during the paroxysms.

During the slow periods extra systoles followed by pauses, more or less fully compensatory, can usually be detected; sometimes they are very frequent, more often only occasional. Single extra systoles are quite common between paroxysms which are of short duration and which follow one another at brief intervals.

During the paroxysms the veins of the neck are prominent, distended, hard and pulsate with great rapidity. Often two pulsations of the jugular may be seen to correspond to each systole of the heart.

In most instances the attacks are not affected by the position assumed by the patient and continue whether he sits up or lies down without change in rate.

When seen only between the attacks the diagnosis rests largely on the history, but the patient's description of the attacks is usually

so clear that there is little difficulty in classifying the abnormal activity.

The cases which present the most obscure diagnostic problems are those with very frequent short paroxysms separated by equally short periods of slow rate broken by frequently occurring extra systoles. These are often wrongly classified as complete irregularity due to auricular fibrillation. They may usually be assigned to their correct category by means of a careful and prolonged study of the ordinary physical signs. Their status may be absolutely settled by graphic records.

The *polygram* brings out clearly some features of the paroxysms which are observed with great difficulty by the ordinary means of eliciting physical signs.

In figures 3 and 4 are shown brachial and jugular tracings taken from a woman 35 years of age. Figure 3 shows the usual condition of her pulse; the rate is 82; the arterial pulse is of good size and well sustained; the jugular pulse shows a normal sequence of waves *a*, *c* and *v*; the *a-c* interval is normal (less than 0.2 second). Figure 4 is a record taken during her second paroxysm, which lasted 2 days without interruption. At the time the tracing was secured the attack has been under way for 24 hours. The heart at this time was beating rhythmically at a rate of 182 per minute. The small volume of the brachial pulse is in great contrast to that of the slow periods. The venous curve shows, in place of the well-defined waves of the slow heart rate, one large wave and one small notch to each cycle. The interpretation is that the auricle and the ventricle are contracting simultaneously, so that the veins are unable to empty into the right auricle in the normal manner. The large jugular waves, much greater than the jugular waves of the normal period, are due to a summation of the *a* and *c* waves. It will also be seen that during the paroxysm the *a-c* interval is considerably prolonged (over 0.3 second), indicating that there is a delay in the conduction of the stimulus from the auricle to the ventricle. This is not an uncommon feature in tachycardias, the excessive functional demand on the slender *A-V* bundle leading to its partial exhaustion.

In these two figures the respiratory curve is brought out in the venous tracing. That in this case the dyspnea was not very marked

is evidenced by the facts that the breathing was 24 during the slow rate and only 30 during the attack, and that the excursion is not very much greater during the paroxysm.

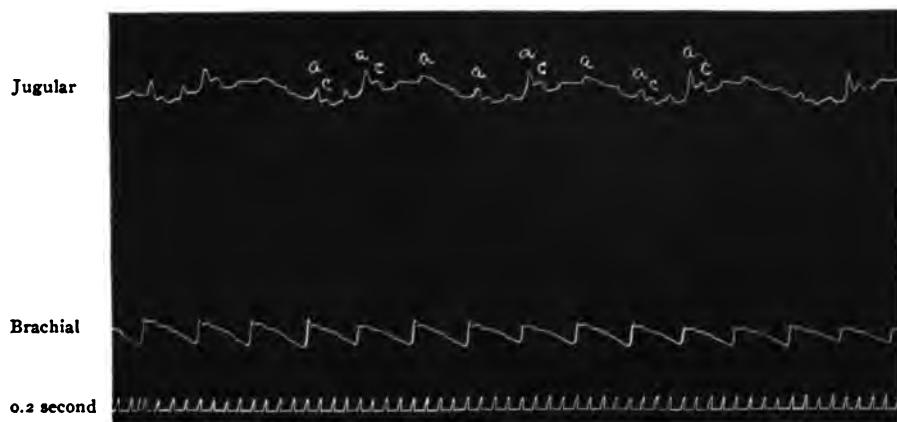


FIG. 3

Normal. Rate 82. Respiratory rate 24. For paroxysm of auricular tachycardia in the same individual see figure 4.

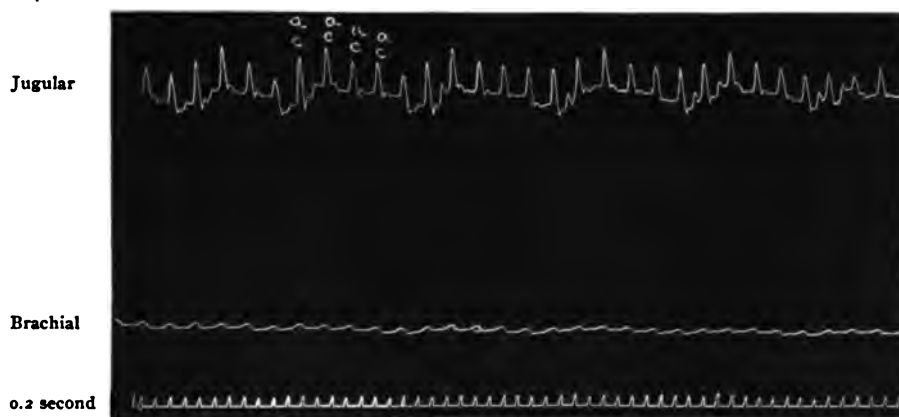


FIG. 4

Auricular tachycardia. Rate 182. Respiratory rate 30. For record of the same case between attacks see figure 3. Compare in the two records the volumes of the arterial and venous pulses. The jugular records are quite different in form, the large wave of the paroxysm = $a + c$.

Tracings from another case of auricular tachycardia are shown in figures 5 and 6. As in the preceding case, the contrasts between the cardiac rates (72 and 174) and the arterial pulse volumes of the two periods are shown in the brachial tracings. During the

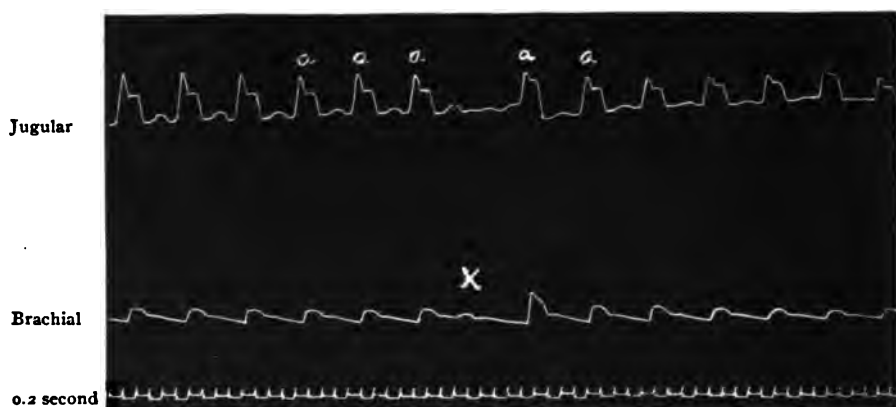


FIG. 5

Rate 72. Between attacks same individual as figure 6. At X is shown an extrasystole with an incomplete compensatory pause. Note the a wave is large and the a-c interval is of normal length.

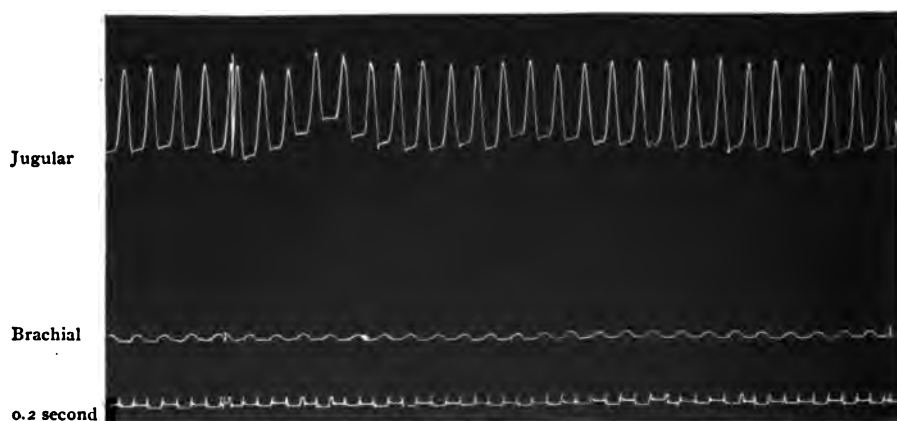


FIG. 6

Auricular tachycardia rate 174. For slow rate see figure 5. The large venous waves are the result of the simultaneous contraction of the auricle and the ventricle. Each one of these waves belongs to two cycles, they are composed of a c wave of a cycle just completed and an a wave of a cycle just beginning. The a-c interval is abnormally long.

paroxysm (figure 6) only one large wave appears in the jugular to each cardiac cycle.

The slow period (figure 5) is interrupted at one point (X) by an extra systole with an incomplete compensatory pause, hence we may conclude that it probably had its origin in the auricular wall. It is a series of such extra systoles which constitute the paroxysm.

Figure 7 was taken from a man of 36 during a prolonged paroxysm. The ventricular rate is 158 and is perfectly rhythmic. The jugular tracing shows the great venous congestion and the very large waves which are due to simultaneous contractions of auricle and ventricle; conduction is delayed. The exact point in the auricle which has become the temporary pace-maker for the whole heart cannot be definitely settled from the polygraphic record. The respiratory curve upon which the large jugular waves are superimposed show that, in spite of the prolonged attack, the breathing is not greatly accelerated; at this time it was 22 to the minute, but quite irregular.

A rare tracing from a case of ventricular tachycardia is reproduced in figure 8: The brachial shows at *A* the usual rate for this patient between attacks (92 per minute). At *X* isolated extra systoles, each with a complete compensatory pause, occur; the premature beats are so weak that they make practically no impression on the brachial pressure. At *B* are shown two short paroxysms of tachycardia, indicating the manner of the abrupt onset and termination of the attacks. The *a-c* interval of the "normal" rhythm of this patient was always longer than that of a normal heart, measuring nearly 0.3 second.

During the paroxysm the auricle contracted in response to the "retrograde stimulus" from the ventricle; this cannot be conclusively made out in the polygram, but is substantiated by electrocardiographic records (see figure 21). The irregularity of this pulse is so extreme that it might easily have been mistaken for a case of "complete irregularity" and auricular fibrillation, had no graphic records been secured.

The *electrocardiogram* gives us information in regard to paroxysmal tachycardia which we can obtain by no other method. Through this agency we have discovered the real mechanism of the attacks. The knowledge acquired in this way tends to emphasize

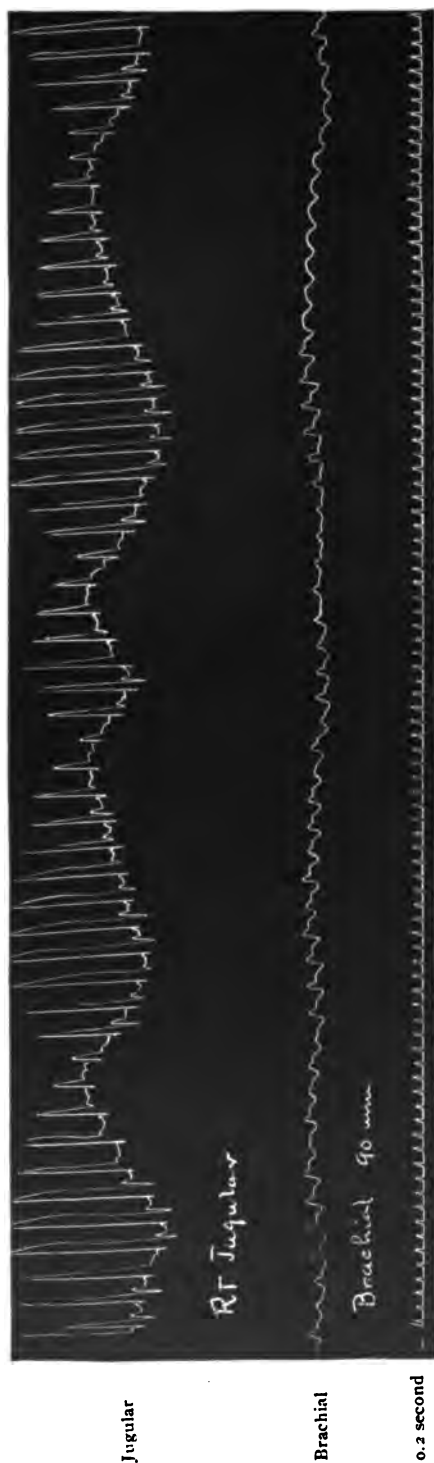


FIG. 7

Auricular tachycardia taken during a paroxysm. Ventricular rate 138. Note the very large venous waves due to venous stasis and the respiratory curve upon which the jugular pulsations are superimposed.



FIG. 8

Ventricular paroxysmal tachycardia. Beginning and termination of attacks. At A' normal rate 92 per minute. At B paroxysms of tachycardia, rate 170 per minute. At X isolated ventricular extrasystoles which show in the jugular but not in the brachial.

the importance of the muscle cell changes and to minimize the rôle played by the extra cardiac nerves in inducing this change in cardiac activity. These graphic records convince us that a new point in the heart wall has become the temporary pace-maker of the heart. The proof is most clearly demonstrated, if we study the records of such a case as is shown in figure 21, where the evidence is complete in a single curve. This is from a case of ventricular paroxysmal tachycardia, a condition of extreme rarity, hence it will be better to first direct our attention to the more common forms, namely, tachycardias of auricular origin.



FIG. 9

Normal. Rate 80. Same case as figure 10. Lead II.

Such a case is illustrated in figure 10, which was taken during a paroxysm in which the heart rate was 167. Figure 9 was secured from the same patient a few hours after the cessation of the attack. This record shows a perfectly normal curve for a heart with a rate of 80. Both records were taken by lead II (right arm and left foot). If we should superimpose the ventricular portion (beginning of *R* to the end of *T*) of the cycles shown in figure 9 on one of the cycles of figure 10, we would find that they correspond in every particular, except that the summit of the *T* wave shows constantly a deep notch. If we compare the records further, we note that in figure 10 there is no wave which corresponds to the well-marked *P* wave of figure 9. Careful measurement shows that the notch in the *T* wave (figure 10) occurs at exactly the time at which a *P* wave of the normal rhythm should precede the *R* wave, hence we conclude that the positive *P* wave of the normal rhythm is replaced by a negative wave notching the *T* wave of the paroxysm.

In studying the auricular extra systole, it was shown that when the premature beat started from a point in the auricle at some distance from the sinus node, the *P* wave of the electrocardiogram was distorted in form, or even completely reversed in direction,



FIG. 10

Taken during a paroxysm, rate 167. Lead II. Same patient as figure 9. Note inversion of *P*, which notches the summit of *T*. Auricular tachycardia. The pacemaker of the heart is in the lower part of the auricle.

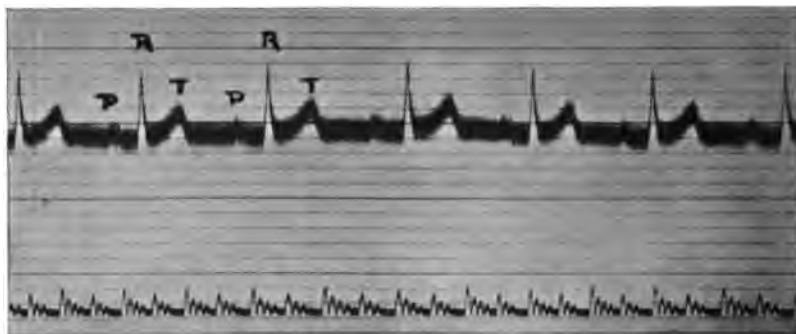


FIG. 11

Same patient as figure 12. Rate 76 between attacks. Taken by lead II. *P* is slightly notched, otherwise the curve is normal.

hence in the records under consideration we are led to conclude that the paroxysm shown in figure 10 is composed of a series of extra systoles having their origin at a point in the auricular wall considerably removed from the site of the normal pace-maker.

Figure 11, taken by lead II, shows a normal electrocardiogram, except for a slight notching of the *P* wave. Figure 12 was taken from the same patient during an attack which lasted for one hour, during which the heart rate was 174. Here the ventricular por-

tions of the two records are almost identical, except that the waves of the paroxysm are a trifle smaller than those of the slow rate. During the paroxysm no *P* wave can be definitely located; in this case it was probably so small that it caused no distortion of the relatively large *T* wave.

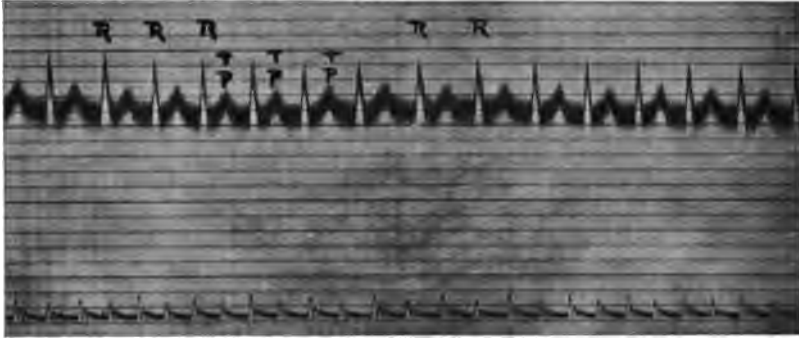


FIG. 12

Auricular paroxysm, rate 174, lead II. From same individual as figure 11. The small *P* wave is submerged in the large *T* wave.

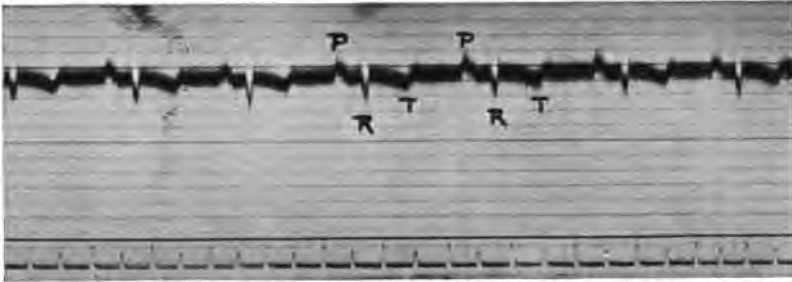


FIG. 13

Slow period, same case as figure 14. Rate 75. Lead III. *P* is notched, *R* and *T* have a downward direction.

Electrocardiograms of another case of auricular paroxysmal tachycardia are shown in figures 13 and 14; both records were taken by lead III (left arm and left leg).

When figure 13 was taken the heart rate was 75 per minute. This record shows several abnormal features; the *P* wave is slightly notched and *R* is directed downward (the latter feature is quite

usual in hypertrophy of the left ventricle), *T* is also directed downward. During the paroxysm (figure 14), the rate is 168. *R* is still directed downward and is increased in amplitude, suggesting a dilatation of the left ventricle. The slow wave between the *R* waves is an algebraic sum of the waves *P* and *T* of the new rhythm.

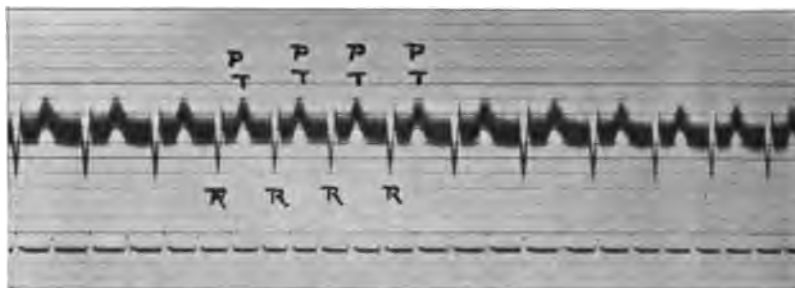


FIG. 14

Auricular tachycardia, rate 168. Lead III. Same patient as figure 13. Ventricular complex increased in size. *T* has become a positive wave. *P* is superimposed on *T*.

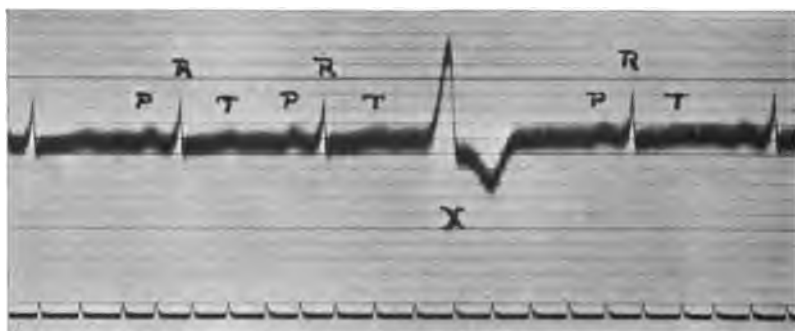


FIG. 15

Rate of 76 interrupted by a ventricular extrasystole. Same individual as figure 16. Lead II.

The next case, illustrated in figures 15 and 16, shows some interesting features, during the slow period (rate 76 per minute), the *P* wave is unusually broad. *R* is slightly notched and the rhythm is broken by an extra systole, which is plainly of ventricular origin. The paroxysm (rate 188) is composed of *R* waves followed by a depression, which in all probability are reversed *P* waves,

having their point of origin in the lower part of the auricle, possibly near the *A-V* node. The *P-R* interval is prolonged, measuring over 0.2 second, exhibiting the delay in conduction which is not an uncommon feature of these cases. In this instance the complexes of the paroxysm probably represent extra systoles of auricular origin and do not conform to the type of the isolated extra systole which interrupts the slow rhythm (figure 15).

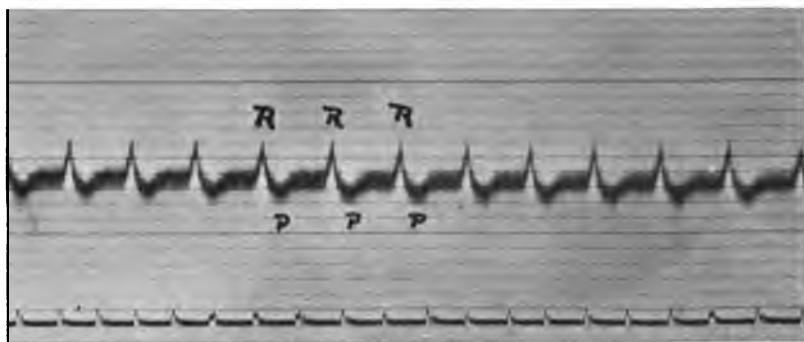


FIG. 16

Paroxysm of tachycardia, rate 188. Lead II. From same case as figure 15. Note reversal of *P* wave and prolonged *P-R* interval. Abnormal pacemaker probably situated near the *A-V* node.

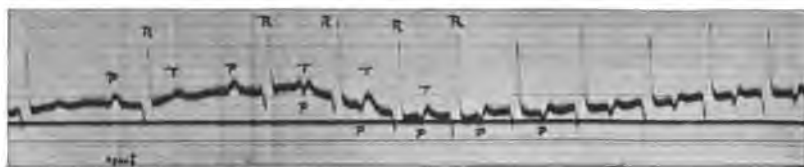


FIG. 17

Transition from slow rate to paroxysm. Note dislocation of pacemaker to a point low down in the auricle as evidenced by the abrupt change in the direction of the *P* wave. The ventricular complex is unchanged except as its contour is broken by the reversed *P* wave. Compare figure 10.

Figures 17, 18 and 19 depict the mode of transition from the slow to the rapid and from the rapid to the slow rate in different cases.

The onset of a paroxysm can be seen in figure 17 and the dislocation of the pace-maker from the sinus node to a point low down in the auricle is indicated by the change in forms of the *P*

wave from a positive to a negative deflection. In the first cycle of the paroxysm the reversed *P* wave falls at the apex of the *T* wave, but subsequently notches the earlier portions of this part of the ventricular complex.



FIG. 18

Abrupt termination of a paroxysm. Transition distorted by extrasystoles of ventricular origin. Pacemaker of paroxysm located near the A-V node. Compare figure 16.

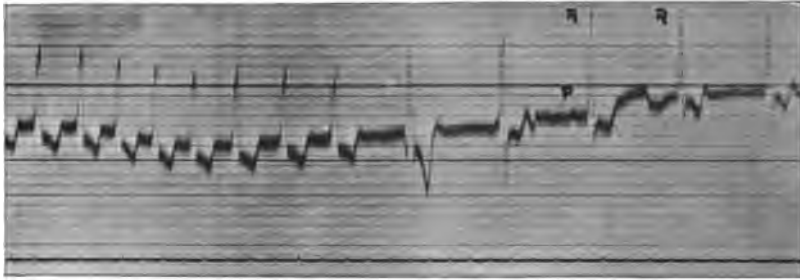


FIG. 19

Auricular tachycardia, rate 160. Transition to rate of 70. Origin low down in the auricle.

The offset of a paroxysm is shown in figure 18. The bizarre complexes which intervene between the paroxysm and the slow rhythm probably represent extra systoles of unusual types, and are doubtless the kind of cardiac activity which give the patient the subjective sensation of "throbs" or "thumps" at the time of the transition.

Another transition from a heart beat of 160 to one of 70 is shown in figure 19. The curve is somewhat distorted by the movements of the patient produced by the sensations experienced at the time of the termination of his attack.

The most convincing evidence of the nature of the mechanism in paroxysmal tachycardia is brought to view when we are fortunate enough to secure in a single record periods of slow rates interrupted by single extra systoles, continued into periods of tachycardia. Such records are shown in figures 20 and 21.

A short paroxysm of tachycardia (rate 168), changing to a slow rate (86) broken by extra systoles, is shown in figure 20. The patient, from whom this curve was taken, was a physician, 65 years of age, in whom the diagnosis of "complete irregularity," due to auricular fibrillation, had been repeatedly made. The correct diagnosis was hardly possible until electrocardiographic records were secured. The slow rate is interrupted by auricular extra systoles (X_1) and another type of extra systole (X_2) which has its origin in the ventricular wall. The auricular premature beats have their origin high up in the auricle, since the P wave of the extra systole is a positive wave, as is shown by the waves which are clearly the sum of T and P . The paroxysm is composed of both kinds of extra systoles, but the auricular type predominates, which is also the case in the period of slower cardiac activity.

The electrocardiogram of a case of ventricular tachycardia¹ is shown in figure 21. Tachycardias of this type are extremely unusual. The bizarre forms of the complexes of his slow rate (80) are seen in the short diastolic (P - T) interval, the broad P wave, the long P - R interval and the unusual form of the R waves. These features alone suggest serious myocardial damage. From time to time there appear isolated ventricular extra systoles (X). The paroxysm (rate 200) is composed of complexes similar in form to those of the isolated extra systoles. Between the large waves of the paroxysmal period are seen small waves (P) which occur with every other cycle. These undoubtedly represent auricular contractions due to retrograde stimuli arising in the ventricle. It appears that every other impulse from the ventricle is blocked. This record conforms in many particulars to the curves obtained experimentally after tying one of the coronary arteries, hence a tentative diagnosis may be made of partial coronary obstruction. The patient is still alive (3 years after the record was taken), hence the diagnosis has not been verified.

¹A complete record of this case will be found in Heart, 1912, Vol. IV, p. 128.

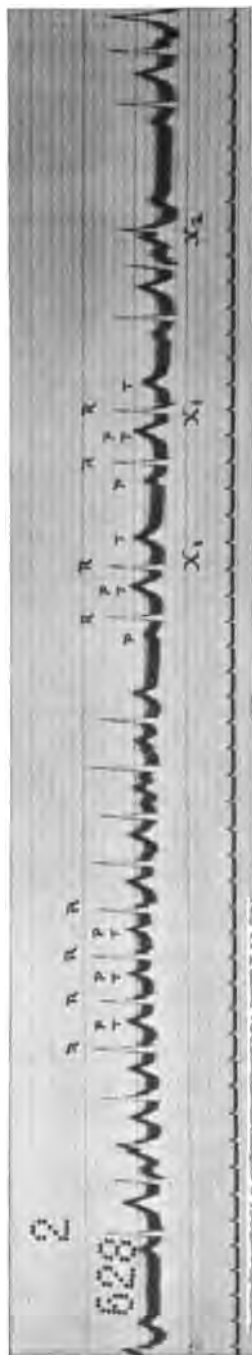


FIG. 20

Paroxysmal tachycardia, rate 168. Transition to slow rate which is interrupted by extrasystoles of two types (X_1 , X_2). The attack is composed of extrasystoles of both types. X_1 are evidently auricular premature beats, X_2 probably have a ventricular origin.



FIG. 21

Ventricular paroxysmal tachycardia, rate 200. The complexes of the slow rate (80) are very abnormal in form. Isolated ventricular extrasystoles at X . Paroxysm made up of complexes of same type as the single ventricular extrasystoles. During tachycardia the auricle responds to every other retrograde stimulus from the ventricle, (P').

CLINICAL SIGNIFICANCE AND PROGNOSIS

There is little doubt that every subject of paroxysmal tachycardia has a defect of the myocardium that must be seriously considered. The prognosis is most difficult. Some patients over a period of many years have attacks which incommode them but little and the attacks become less severe, less alarming, and in some instances disappear altogether. Some have only a few attacks before the fatal termination.

I have never seen a case that was fatal until a number of attacks had occurred, nor have I found such a case reported in the literature.

The condition of the heart in the intervals between attacks is important as an aid in determining the seriousness in the individual case. If at these times the heart shows no abnormality other than occasional extra systoles, one can be reasonably sure that there is no imminent danger. If, however, marked valvular defects are present, if there is evidence of an old inflammation of the pericardium, if there is a general arteriosclerosis, if extra systoles constantly occur at very frequent intervals, and if the heart is embarrassed in maintaining an adequate circulation in the periods of slow rate, the paroxysms will rightly be viewed with much apprehension. The paroxysm is very exhausting to the heart. If the myocardial damage is made evident by the attacks only, the heart will probably successfully carry this stress; if, however, other evidences of myocardial damage exist, the strain of the paroxysm is a far more serious matter. The patients who do particularly well are young subjects with no evidence of cardiac abnormality between attacks. Middle-aged and elderly individuals sooner or later invariably develop other evidences of myocardial insufficiency and, while they may have many and frequent attacks of tachycardia without serious manifestations, the ultimate outlook is less favorable.

The frequency and duration of the individual attacks do not seem to be very important factors in determining the prognosis. Much more important is the severity of the attacks as estimated by the degree of circulatory embarrassment, cardiac dilatation, the congestion of lungs and liver and edema of the extremities. Attacks associated with unconsciousness should be viewed with gravity.

With a history of a moderate number of attacks over a number of years in a young adult, with intervening periods of normal heart action, one may usually give a good prognosis. When the patient is more advanced in years and has paroxysms of increasing frequency and severity, and intermediate periods characterized by signs of cardiac insufficiency, the outcome of any particular attack is doubtful, the prognosis for the future is not good.

PITUITARY EXTRACT—ITS VALUE IN DISTINGUISHING BETWEEN FALSE AND TRUE LABOR PAINS

By SAMUEL WYLLIS BANDLER

Adjunct Professor of Gynecology, New York Post-Graduate Medical School and Hospital

New York

Just why labor occurs on the two hundred and eightieth day is not generally known, except that it is a characteristic of the human species. What the elements are, or whence they come which produce labor pains and the expulsion of the fetus, can only be suspected on the theory that the action of pituitary extract in increasing labor pains points to the hypophysis as the source of the secretion which produces this phenomenon.

One of the greatest aids in labor is the use of pituitary extract given by the hypodermatic method. All that this secretion does, if given in not too large doses, is to increase the labor pains, not alone subjectively but objectively, that is, the labor pains are made effectual, the contractions of the uterus do excellent work. This holds good in the first stage as well as in the second stage of labor. In fact, with the regular use of small doses of pituitary extract in the second stage of labor, with the head well molded through the brim, with the cervix dilated, the use of forceps is diminished almost to nullity.

Pituitary extract, when given before labor pains come on, that is, before the patient is in labor, has no effect whatsoever. It does, as in Caesarean section, contract the uterus, which is of great value, after the fetus is expelled. It does in other cases, such as abortion or miscarriage, have a slow effect in contract-

ing the uterus, but, in the pregnant woman, unless she is in labor, it has absolutely no effect whatsoever in bringing on rhythmical labor pains.

These facts suggested the use of pituitary extract by the hypodermic needle as a diagnostic procedure; in other words, patients often, at or about the expected time, or even a month before, have what are known as suggestive pains. Very often these are only what the text-books call "false pains."

The obstetrician is in doubt as to whether the patient is going into labor. These pains may come, last for a few minutes or an hour or two, and then stop for a day or a week, come on again, and in that way keep the physician on the anxious seat.

Knowing the effect of pituitary extract when the patient is actually in labor, I tried the use of this drug in these cases where we were in doubt, with the idea of determining whether the patient's pains were real or false, and to my agreeable surprise it has proved to be of the greatest value in several instances.

Before reciting any individual cases, I may say that if the patient have pains of this indefinite sort, and a third of an ampoule of pituitary extract be given by needle, if in half an hour another third of an ampoule be given by needle, and if in another half-hour a third of an ampoule be given, and then no regular rhythmic pains come on, the patient is not in labor.

On the other hand, one is surprised to find how often this treatment brings on regular rhythmic labor pains, and the patient goes on through her labor as she would have done under ordinary circumstances, even if no preliminary pains of lesser or greater character had taken place.

Let me instance the case of a patient who went a week over her expected labor period. She had had, on two occasions, pains every ten or fifteen minutes lasting for an hour or two. These pains would then cease.

I determined, on the occurrence of the next so-called irregular pains, to try the use of pituitary extract. When the pains came on every ten or fifteen minutes, the nurse notified me, and when I reached the house after the lapse of an hour and a half all these pains had stopped. I then administered a third of an ampoule of pituitary extract, in half an hour I gave another third, in an-

other half hour another third. The patient went on into regular labor and in six hours was, under chloroform, delivered. This was a primipara.

Another patient, who had had one child, was warned by me to inform me of the first pains she had or of any flow of liquor amnii, or of any signs of blood. She notified me at seven-thirty in the evening that on the toilet she had noticed a slight stain of blood. I immediately went to the house and found the patient fully dressed ready to go out. I said I shall try and see if you are in labor or not. I gave her a third of an ampoule of pituitary extract and in ten minutes the pains came on regularly. She was undressed and put to bed, in half an hour she received a second ampoule, in a half hour a third of an ampoule. Two hours after I entered the house her baby was born. A patient pregnant for the second time, complained during the last week of irregular pains, lasting for an hour or two on several occasions. I finally told her that she must notify me when the next pains occurred. On a Sunday morning, at seven o'clock, the nurse rang me up and told me that the patient was again having the so-called irregular pains. I reached the house at nine o'clock, gave her a third of an ampoule, after half an hour another third of an ampoule. She went on into regular rhythmic pains, the pituitrin was repeated on several occasions, and four hours after I reached the house her baby was born under chloroform anesthesia.

The next is a case of a patient who had had two children, the second one being born in a rather rapid time. I warned her to let me know of the approach of labor pains immediately as they occurred. She rang me up early in the morning. (She lived an hour by automobile from my office.) I immediately went to the house, and when I reached there the patient was almost ready to go to sleep. Through the experience gained from the other cases, I determined to find out whether the patient was or was not in labor. Again, I gave this patient a third of an ampoule of pituitary extract and pains came on. In half an hour I gave her another third of an ampoule, in a third half-hour another third of an ampoule, and in three hours this patient too was delivered under chloroform. Had no pituitrin been given, no one knows

how many hours we might have been waiting for the regular onset of labor pains.

Let me instance the case of another patient who was to be confined in the month of December, about the twentieth. She was very large, I thought because of a great deal of liquor amnii; the patient herself, for some reason or other, felt that a mistake had been made in our calculations. On November the twenty-fifth, on a Sunday morning, she notified me that she was having regular pains at intervals of fifteen minutes. She lived very near the hospital, so I told her to go there and send for her nurse.

When I reached there, the uterus did show every fifteen minutes fairly firm contractions. I gave her a third of an ampoule of pituitary extract, repeated the same every half hour for six doses. The pains continued for a while at intervals of fifteen to five minutes, and then completely stopped. The patient went to sleep. That very afternoon I sent her home and told her she was not in labor. Just four weeks afterward she had the same sort of pains. I was sent for and gave her the pituitary extract in the same manner. She went into a regular labor, and in three hours she was delivered of her infant.

As a final case, let me note a patient who, again like the previous one, was extremely large. I thought because of a great deal of liquor amnii. The patient herself expected the possibility of twins, or the possibility that a mistake of a month had been made in our calculations.

On two occasions, one exactly four weeks before the time at which I had fixed the labor, another a week after, the patient notified me that pains were coming on every fifteen minutes with a sense of pressure on the bladder. The first time I gave her five doses of a third of an ampoule of pituitary extract and the pains completely died away. On the second occasion, I did the same, staying there five hours, giving her six doses of pituitary extract a third of an ampoule each, and again for the second time the pains died down. In each instance I had no hesitation in leaving the patient immediately after the effect of the pituitary extract showed that no labor was going on. When in this last case the labor is to occur I do not know, as the time which I have fixed has not yet been reached.

All these factors, aside from theoretical considerations, show me that in pituitary extract we have a very valuable drug from the standpoint of diagnosis.

I might summarize by saying that any patient who has irregular labor pains, or what are known as "false pains," or thinks she is in labor, who has any symptoms resembling the symptoms she experienced in any of her previous labors, who does not respond to frequent doses of pituitary extract, who does not go into labor after a few such doses have been given hypodermatically, is not at that time in labor.

Of course, this does not aid us in fixing the exact time at which the real labor pains will come on, but it shows us that that particular time is not the time when we are supposed to stand by and await, for hours, the birth of an infant.

PERVERSITIES OF THE INTERNAL SECRETIONS IN THEIR BEARING UPON ORAL PATHOLOGY

By HEINRICH STERN

New York

Experimental and clinical proof is not wanting that some of the internal secretions contribute toward the proper development of structures and contents of the buccal cavity, and that certain perversities of these secretions are liable to announce themselves by maldevelopment or anomalies of the textures of the mouth, especially its bony framework and the teeth.

The definitely known effects of the internal secretions upon the normal development of the bony structures of the mouth are not many, but the demonstrable influences of the pathology of the internal secretions upon the oral structures are considerably less. It should, furthermore, be remembered that animal experimentation can never exactly reflect a certain disease condition in the human organism, and that it may even lead to quite deceptive results and misinterpretations. With this proviso in mind, the following cursory review of the known effects of the internal secretions upon the anatomy and pathology of the jaws and teeth should be interpreted.

The principal glands in question are the thyroid, the parathyroids, the thymus, the pituitary, the ovaries and the testicles.

THYROID GLAND

In endemic goitre and myxedema, disturbances of thyroid function, the osseous growth is more or less interfered with. In persons thus affected, the teeth are poorly developed, and the eruption of the permanent teeth is retarded as the epithelium covering the surface of the dental groove and the dental papilla, the two elements from which the dental textures are derived, are underdeveloped.

PARATHYROID GLANDULES

The parathyroid secretion is supposed to exert a detoxicating activity. Others ascribe to the parathyroids a certain regulatory influence of calcium metabolism. As far as alterations of the dental structures after removal of the parathyroids are concerned, our knowledge is wholly obtained from observations made on animals deprived of these organs. In the incisors of parathyrosectomized rats, alterations in the enamel and dentin, due to calcium deficiency, were demonstrated. The pathological changes consisted in enamel lacunæ and the deposition therein of exposed dentin.

THYMUS GLAND

There is little doubt that the thymus gland exerts certain effects upon the development of the teeth and jaws. In animals whose thymus has been removed, there is a weaker development of the skull, dentition is retarded, and there is marked textural deficiency of the teeth. There is also ample clinical evidence that an insufficiency of the thymus gland has its bearings upon dentition in a pathological sense.

PITUITARY GLAND

Pituitary oversecretion induces changes in the bony framework of the oral cavity, particularly the inferior maxilla and the zygoma. Diminished function of the hypophysis, on the other hand, inhibits osseous growth, retards dentition, and prevents proper structural development of the teeth.

SEXUAL GLANDS

Nothing seems to have been published concerning a direct influence of the internal secretions of the ovaries and testicles on the development of the human teeth. We know, however, that the teeth of castrated animals, especially those of the males, are more or less underdeveloped. This is especially evident in the male hog.

It would be mere speculation to adduce an influence of any of the other glands with an internal secretion (including the adrenals) upon the growth and further development of the jaws and teeth. Such influences, of course, may exist. Yet there is no proof thereof. A single coincidence does not establish a fact any more than one swallow will make a summer.

Some of the secretions of the glands aforementioned may be correlated in their function and may exert *positive* influences; other secretions may also operate unitedly, but may yield *negative, neutralizing, detoxicating* effects. The normal activity of the one set of glands is just as essential to the organism as that of the other. Still, definite conclusions as to the synergism and the antagonism of the various internal secretions, in particular of the few which are supposed to preside over the growth and development of the osseous structures and the teeth, cannot be drawn at this early date.

Ferdinand Blum, one of the first investigators of thyroid function, maintained, and I believe still maintains, that thyroid activity is wholly or in the main one of detoxication. His contention is practically disproved, and it is the consensus of opinion of most experimenters and clinicians that it is a positive influence which is yielded by the secretion of the thyroid gland. Substantially the reverse has taken place in the view held as regards the quality of the effect of the internal secretion of the ovary. While it was generally understood that the ovary was a gland with a positive internal secretion, Okintschitz has very recently demonstrated (Archiv f. Gynäkologie, Vol. CII, No. 2) that the corpus luteum gives off a negative internal secretion, one which neutralizes the toxic substances circulating in the organism. In accordance with Okintschitz's findings, the influence of the internal secretion of the ovary upon the growth and development of the teeth and the bony framework of the buccal cavity is entirely negative in nature; it is in no way or manner directly concerned in the upbuild of the

osseous structures, including the teeth, and it is only of import in the neutralization of certain toxic elements which may interfere with the function of the glands presiding over calcium metabolism.

Besides the organs that are known to exhibit an internal secretion, there are undoubtedly many others possessing the same quality, in which, however, this has as yet not been demonstrated. This also pertains in all probability to some of the organs whose internal secretions directly or indirectly contribute toward the development and maintenance of the manifold structures in the buccal cavity. Moreover, the physiological activity of certain of the organs furnishing an internal secretion is of different intensity in the various phases of life. It is not so long ago, for instance, that the opinion was held that the thymus gland was completely vestigial in the adult. While the deterioration of this gland is by no means so consummate in mature age as was once supposed, there cannot be any doubt that the import of the thymus to the infant below two years of age is greater than to the older child, inasmuch as this organ does no longer increase after that age and that it actually decreases in size after puberty. In conformity with the larger volume of the gland stands also its physiological activity.

The activity of a certain gland with an internal secretion may in a sense become a vicarious one. That is, the deficient function of one or more glands may be compensated for by a correlated force issued by another gland. It stands to reason that this pertains primarily to the glands yielding secretions of similar quality, i.e., secretions that would normally display synergistic activity.

Again, at different ages the activity of one or the other of the glands predominates. As far as the subject under consideration is concerned, we have seen that at a very early age it is the function of the thymus which prevails. A few years before puberty, thyroid activity, if not at its height, is very intense, and presumably extends its influence to the oral cavity and its contents, and after puberty it is the sexual glands which slowly ascend to their highest attainable degree of physiological development. While the activity of the secretions of these glands differs widely, that is, while one of these secretions can never fully replace the other, the predominance of each of them at the proper period of life is a physiological phenomenon.

The development of the osseous structures of the organism, necessarily preceding that of the higher specialized tissues, demands the full activity of the thymus and all the glands with correlated function. The more specialized textures pre-eminently require for their growth and proficiency the synergistic secretions of the thyroid and other glands. When maturity is reached, direct thyroid activity, though essential, should normally become reduced in intensity, while, for the time being, the function of the sexual glands are more or less displayed. This explanation, I know, is based on rather a teleological reasoning, but I believe that it assists in the understanding of the argument.

Glancing over the brief résumé of the facts concerning the influence of the glands with an internal secretion upon the structures of and in the buccal cavity, it appears as if nothing were known about their effect on structures other than osseous or dental. This, however, is not the case, as there are at least two affections of the soft tissues of the oral and contiguous cavities which may be the results of deficient thyroid activity.

One of the soft tissue involvements is evidenced by edematous swellings of the visible mucous membranes of the tongue, uvula and nose. Sir Felix Semon states that the laryngologist is occasionally consulted on account of stoppage of the nose, accumulation of mucus in the posterior nasal cavity and the throat, a feeling as if the tongue were too large for the mouth, on account of retarded articulation in speaking and the leathery, dull sound of the voice, and that a more minute examination will often demonstrate the myxedematous nature of the various disturbances.

The other manifestation of the influence of an internal secretion upon non-osseous structures of the mouth is noticeable in the gums. It is also due to thyroid deficiency. I was the first to describe this phenomenon as a possible symptom of hypothyrosis. (Further Experiences with Thyroid Modification and Therapy, Am. Med., March, 1912.)

My observation consists simply in the fact that *in a certain proportion of gingival lesions these are a part manifestation of myxedema or minor degrees of hypothyrosis, and that these lesions disappear together with the other phenomena of thyroid insufficiency on the introduction of thyroid therapy.*

The possible etiologic connection between deficient or perverse thyroid function and Fauchard's or Riggs' disease was observed by me as far back as 1902, when, on the occasion of a series of lectures on the disorders of catabolism to post-graduates in medicine, I maintained that, besides local causes and the usually assigned systemic affections as diabetes, gout, rhachitis, leukemia, arteriosclerosis, etc., a hypothyrosis may stand at the foundation of the gingival process. I have held this view ever since, no matter to what fanciful causes others have tried to fasten inflammations of the gingivæ. The discovery of the ameba buccalis even could not prompt me to change my views in respect to the possible hypothyrotoxic origin of a certain number of instances of gingivitis.

(That amebæ can be demonstrated in almost every mouth at almost any time, and this in spite of scrupulous cleanliness, is known for a number of years, but that they are pathogenic and cause gingival disease or pyorrhea has not been proved to my satisfaction. Neither is there a scintilla of proof that the hypodermatic administration of emetine, or the local use of this drug or its mother substance, ipecac, can per se cure these affections.)

Of course, there are local conditions which favor the production of the gingival process, or aggravate it in case it is already existent; and there are systemic affections, especially syphilis, which may be accompanied by Riggs' disease. Many cases of syphilis, however, do not exhibit a gingival lesion, and it is an open question whether, when it has ensued, it is of syphilitic or mercurial origin. Again, I am convinced that many instances generally spoken of as Riggs' disease of syphilitic causation, in reality are not cases of this affection at all, but the end-results of mercurial stomatitis.

The alleviation of one of the constitutional diseases, as for instance the suppression of hyperglycemia, is hardly ever followed by a prompt and well-marked improvement of the gingival condition. When, on the other hand, the affection of the gum tissues is of hypothyroid origin, it disappears, as a rule, synchronously with the other hypothyroid manifestations. While, therefore, a causal relationship between the constitutional and local disease is by no means a certainty, the dependence of the gingivitis upon the thyroid deficiency is definitely established.

There are, hence, anomalies of the gingivæ which are part and

parcel of a hypothyrotoxicosis. If this yields to the exhibition of thyroid, the gingival phenomenon will also accede to it. (In a majority of the pertaining cases it is, nevertheless, essential that the teeth and gums be kept in as healthy a condition as possible, that the tartar be removed and local treatment instituted when the circumstances call for it. At the same time, reliance must not solely be placed upon the removal of possible local irritants or defects.)

In the article already mentioned, I quoted from the records of 52 cases of hypothyroidism which were under my continued observation for from two months to nearly two years. Of these 52 cases, 28 showed no gingival symptoms at all; in 10 cases there existed mild affections of the gums (not of a pyorrheal nature, and unaccompanied by atrophy, etc.), while in the remaining 14 cases there had ensued more or less pronounced gingival manifestations. Though these 14 cases had received more or less local care at the hands of dentists, the results therefrom were, generally speaking, indifferent. The administration of from 15 to 45 centigrams (3 to 9 grains) of thyroid for from six to fourteen weeks was followed by a complete cure of the gingival process—for the time being—in 3 instances, a distinct improvement in 5 others, and a slight improvement in an additional 2 instances. The remaining 4 cases were not ameliorated at all after from three to four months' administration of the drug. One case of the last group, however, became markedly better when a second attempt at thyroid compensation was undertaken some time later. The fact that out of 14 cases of hyperthyrosis with gingival symptoms 10 were beneficially influenced, shows conclusively that these manifestations were due to an insufficient thyroid secretion, and that thyroid therapy furnished the compensating factor.

Since reporting my observations three years ago, I have seen a very large number of persons in a hypothyroid state. In a certain proportion of the cases, it was the pathological condition of the gums which prompted me to search for other possible manifestations of hypothyroidism. Gingival disease of manifold type and degree was encountered by me in fully twenty-five per cent. of all the instances of deficient thyroid activity. These gingival affections seemed not to be of hypothyroid origin in about three or four per cent. of the cases, that is, no improvement of the local

pathological state ensued with or after the abatement of the other hypothyroid phenomena.

Specifically, the gum affections in hypothyroidism that responded to thyroid therapy could be differentiated as slight localized reddening, simple gingivitis with or without pericementitis, marginal gingivitis, spongy gums, lacerated gums, deeply seated gingivitis and pyorrhea alveolaris. The teeth were more or less loosened in a goodly proportion of the cases.

The majority of the gingival affections were found between the thirtieth and fiftieth year of life. There were about three times more women than men affected with disease of the gums. The preponderating number of women had either never been pregnant or had not borne children for a long time. The hypothyroid state varied from languor, the falling out of hair and a fleeting edema to adiposis dolorosa, or completely developed myxedema.

In all instances of hypothyrosis, thyroid administration must be continued for protracted periods. When improvement has supervened, the medication may be entirely stopped or the dosage and the frequency of its exhibition may be diminished for some time. It stands to reason that the gingival process may again manifest itself together with a recrudescence hypothyroid state. For this reason we can only speak of a cure of hypothyroid gingivitis in the same sense as we speak of a cure of the hypothyrotoxicosis itself.

In hypothyrotoxicosis it is especially the peripheral organs that are most affected. Denutritional and degenerative changes take place more readily in peripheral than deeper seated or central parts, for the reason that the terminal arterioles and their nerve supply are prone to be affected by even comparatively slight untoward influences. The smooth and vascular mucous membrane covering the gums is a component part of the internal integument. Together with the fibrous tissue which is intimately connected to the alveolar periosteum, it forms a peripheral structure that is readily susceptible to systemic influences of almost every kind. Denutrition, disease and structural degeneration will be the natural and ultimate result.

ACUTE MYELOID (MYELOBLAST) LEUKEMIA

By ARTHUR F. BEIFELD

Instructor in Medicine, Northwestern University Medical School
Chicago

The development of our present knowledge of the leukemias took place in several well-defined stages. Virchow, in 1845, placed the condition upon a firm pathologic basis by differentiating it from pyemia. Ebstein¹ (1889) and Fraenkel² (1895) recognized and defined, clinically and hematologically, respectively, an acute leukemia. At first, all acute leukemias were looked upon as lymphatic; in fact, as recently as 1907, Naegeli³ regarded only eleven cases in the literature as unambiguously myeloid. In the past decade many cases have been shown beyond question to be myeloid, by means, particularly, of careful histologic studies and modern staining methods; the case of Schultze⁴ stands out as one of the first proved instances of an acute myeloid leukemia. Opinion, to-day, seems to incline toward the position that the myeloid is much more frequent than the lymphatic (Türk,⁵ Ziegler and Jochmann,⁶ Jochmann and Blühdorn⁷).

The recognition of an acute leukemia, disregarding for a moment the type, is not difficult. The acute forms are unquestionably commoner than the chronic; in the absence of histologic examinations and hematologic studies, however, they pass under the diagnosis of morbus maculosus (Werlhof), scorbutus, ulcerative angina, sepsis, pernicious and other high-grade anemias.

More difficult is the differentiation of the types of acute leukemia. Clinically they parallel one another closely. The *bête noire* is the presence in the blood in both forms, often predominatingly, of large mononuclear cells, with more or less basophilic cytoplasm, and without granules. To determine whether these cells are of lymphatic or myeloid origin, in the particular case, may be impossible, despite the employment of refined morphologic, chemical and biologic criteria. In certain cases the autopsy is necessary for a final opinion (case of Herz,⁸ which the writer saw); in others the diagnosis can be made with a fair degree of certainty from the blood picture; while, in a small proportion, the intra vitam diagnosis is unusually clear. The following case belongs to the latter category:

K. C., Russian, twenty years old, a laborer by occupation, entered the Cook County Hospital November 16, 1914. Important light is thrown upon the possible duration of the case by the fact that he had passed the inspection of the immigration authorities eight months before. Except for vague abdominal symptoms (constipation) he had always been well up to the onset of the present illness, four months before. At first he had experienced cramp-like abdominal pains, had lost his appetite, and was troubled with headache and vertigo. Gradually his condition became worse, until two weeks ago increasing weakness forced him to go to bed. The last fortnight had been marked by great loss of strength, swelling of the feet, dizziness, constant headache, dyspnea, constipation, abdominal pain and vomiting, repeated epistaxis and bloody stools.

He admitted the moderate use of beer and whiskey and denied venereal infection.

The young man was extremely anemic—integument, mucous membranes, lobes of the ear—the skin presenting a rather lemon-yellow tint. His general nutrition was good. The physical examination revealed little: clotted blood in the nasal passages and nasopharynx; *no ulcerative lesions* in the mouth, about the teeth, on the tonsils, or in the pharynx; a systolic murmur over the entire precordium, soft in character, with normal heart borders; a spleen, slightly enlarged on percussion, the edge of which could readily be palpated on deep inspiration; a just palpable liver edge; a few small, discrete, not tender lymph nodes in the left cervical chain and in the left axillary, left epitrochlear and both inguinal groups; tenderness over the cranium, sternum, tibiae and femurs; and a number of quarter-size deep hemorrhages over the tibial surfaces of both extremities.

There was observed, in addition, bilateral retinal hemorrhages, an irregular low-grade temperature, and a blood pressure of 112 mm. systolic and 60 mm. diastolic.

The condition of the patient remained practically unchanged, except for progressive weakness and lethargy, until his death on November 28th, twelve days after admission.

The enumeration of the erythrocytes and leukocytes and the estimations of the hemoglobin (Dare) are shown in tabular form below:

	11/18	11/21	11/21	11/23	11/24	11/26	11/27
Erythrocytes	952000	768000	776000	670000	592000	672000	648000
Leukocytes	24850	40000	46900	41650	45000	71200	82400
Hemoglobin	12%	16%	14%	13%	13%	11%	11%

The high-grade and progressive anemia evident from the foregoing is not uncommon in the acute leukemias, which as a rule affect the erythropoietic system far more vigorously than do the chronic forms. Not infrequent, further, is a high-color index, in this case averaging slightly greater than one. The good state of nutrition, the pallor, the marked oligocythemia, and the high-color index suggest pernicious anemia, but the leukocytic increase—foreign to P. A., except as a terminal septic episode—and the qualitative study of the white cells, particularly the presence of myeloblasts and their derivatives, speak against the Biermer type of anemia.

The submyelemic value—24850—present on admission, answers one of the clinical criteria of an acute leukemia, namely, that a case must be under observation at a time when leukemic values have not developed, thus eliminating the possibility of an acute exacerbation of a chronic form, or the influence of a terminal septic event. The increase in the course of ten days to a more nearly leukemic value—82400—points also to the existence of a frankly acute case and its development under observation.

The leukocytes were distributed as follows:

	Myelo- blasts	Premyelo- cytes	Myelo- cytes	Polynuclear Neutro- philes	Lympho- cytes	Türk's Irrita- tion Forms	Normo- blasts per 100 cells counted
First Count	62.6%	9.9%	0.5%	21.1%	5.8%	0.1%	More than 6
Second Count	56.7	10.7	2.0	23.6	7.0	0.0	5
Third Count	57.0	21.0	1.6	14.4	5.8	0.2	2

The first and second enumeration, it will be noted, show no appreciable difference. The last count, made from blood taken shortly before death, differs strikingly, in two particulars, from the foregoing ones. In the last count the premyelocytes—cells with myelocytic nucleus and beginning granulation—have doubled in percentage

as compared with the first two examinations, and this it will be observed is at the expense of the polynuclear neutrophils. In other words, if, in each count, premyelocytes and polynuclear neutrophils are added, the total is practically the same in all three.

This variation in the final count might be attributed to functional (chemical) variations in the cells themselves, with a resulting greater affinity of the granules for the stains, were it not for the fact already emphasized that the increase in granular mononuclears is at the expense of the polymorphonuclears. A more reasonable explanation for the variation, then, would be that as death approached the myeloid tissue became more exhausted, producing in place of mature cells, their forerunners, the premyelocytes. In keeping with this theory is the gradual diminution of nucleated red cells.

That the case under consideration is one of acute leukemia cannot be doubted in view of the course, the hemorrhagic diathesis, the development under observation of a leukemic from a subleukemic state, the high-grade anemia and the predominance of large cells. Points speaking for the myeloid origin of the case are these:

1. The striking atypicalness of the white cells, suggestive always of myeloid upheaval. Pertinent details of the blood picture will be considered below.

2. The unusually marked evidence of *transition from undifferentiated mother cells to cells of the myelocytic row*. This is particularly well shown in the last differential count, in which 21 per cent. of the cells counted represent transitions from the non-granular to the granular condition. Apart from histologic studies of the tissues involved, this is unquestionably the most satisfactory means of separating the two types of acute leukemia (Naegeli, Türk). In the lymphatic form, the large mononuclear undifferentiated cells show their lymphoblastic origin in that the cells about them are maturer, or ripe, lymphocytes, and myelocytes are few; while, in the myeloid type, apparently similar large cells are seen to have as descendants granular forms, that is, premyelocytes and myelocytes.

3. The Winkler oxydase reaction. Recourse has also been taken to chemical means to differentiate the acute leukemias. Based upon the hypothesis that cells of myeloid origin contain an oxidizing ferment-oxydase, which cells of lymphatic origin do not, several

tests have been formulated for the recognition of this ferment. The most satisfactory of these seems to be the indophenol reaction of Winkler,⁹ the application of which to the leukemias was made by Schultze.¹⁰ The reagents, a 1 per cent. aqueous solution of alpha naphthol (to which an equal weight of sod. carbonate has been added to promote solution), and a 1 per cent. aqueous solution of dimethyl-phenylendiamin, if brought together in the presence of oxygen, produce a blue pigment, the exact composition of which is uncertain. Frozen sections from fresh tissues or from organs which have stood for months in formalin, and blood-smears hardened in formalin or alcohol, if treated successively with the two reagents, present blue granules, usually fine, sometimes coarse as a result of coalescence, corresponding to the oxydases present in the cell (not to the granules of the Ehrlich school). This color phenomenon is transient, fading in several hours. Previous heating of a specimen destroys the ferment; alcohol causes the color to disappear, though it can be restored by renewed application of the reagents.

The indophenol reaction, in our case, was exquisitely positive. Nearly every cell showed itself to be of the myeloid system by the appearance of numerous blue granules. As a control, use was made of a blood-smear from a case of acute lymphatic leukemia recently under observation; only here and there did a cell show the oxydase granules (polynuclears).

4. Morphologic differentiation of myeloblast and lymphoblast. Oftentimes the usual criteria available here are of no value. Myeloblasts are, on the average, larger than lymphoblasts. In our case, as will be detailed later, are many micromyeloblasts, thus obscuring the value of size standard. No special stains were employed to demonstrate the number of nucleoli or Schridde's perinuclear zone. Speaking directly, however, for myeloblast as against lymphoblast are the delicacy of the nuclear chromatin—without the thickenings seen in lymphocytic cells—the presence in many cells of one or more deep clefts (Rieder type) and the tendency of the nucleus in many of the small and medium-sized cells completely to fill the cell body (Pappenheim)¹¹.

Of the foregoing features, that which speaks directly and unequivocally for the myeloid nature of this leukemia is the unusually clear evidence that the descendants of the large mononuclear un-

granulated cells are cells of the myelocytic type. Concerning the value of this manifestation hematologists are generally agreed, whereas in the case of other criteria of difference—morphology, oxydase reaction, etc.—there is a considerable variation of opinion.

It seems worth while to enter somewhat in detail into the morphologic characteristics of certain of the white cells. The predominant type—the myeloblast—appears in many forms, this in itself speaking for a tumultuous myeloid activity. Most numerous is the familiar type, usually considerably larger than the myelocyte, with leptochromatic nucleus, several nucleoli, a considerable cytoplasm of various degrees of basophilic intensity, containing no granules, and often showing one or more vacuoles in the cytoplasm. There is, further, a considerable percentage of cells about the size of the myelocyte, with a nucleus identical with that of the myeloblast and the most slender of cytoplasmic zones or none at all. These cells often present a deep cleft, or several clefts, which may divide the nucleus into two parts. These medium-sized cells, but more particularly another cell also numerically high, roughly the size of a normal lymphocyte, suggest very strongly the lymphocytic character. A considerable discussion has been engendered as to the origin of these cells. We have seen fit to class them as meso- and micro-myeloblasts on the basis of criteria set up by Pappenheim, namely, the nuclear character—delicate chromatin as compared with the cruder structure of the lymphatic cells—which present chromatin knobs or heaps—the absence of protoplasmic zones, this being particularly significant in the case of the meso-myeloblasts, and the deep clefts already described, also significant of myeloid origin, especially in cells of medium size. Finally, in some of the cells in which the protoplasm is somewhat more abundant, granules, usually few in number, can be found. (Lydtin¹² and others describe cases of acute micromyeloblastic leukemia.)

The polynuclear neutrophiles in all specimens are atypical in point of size—nearly all are large—and in the absence, in the majority, of granules. To this latter feature Naegeli¹³ calls attention in the second edition of his book.

No basophilic or eosinophilic cells are present. This is the rule in the acute form of myeloid leukemia. Nor can any cell be definitely identified as a normal large mononuclear (transitional).

A few of the cells contain scattered granules suggestive of azur granules (Wright stain). There are rarely more than five or six of these granules in a cell, often only one or two. Some are three or four times the size of the usual eosinophilic granule. All possess a vivid, almost cherry-red color not at all suggestive of eosinophilic granules. Azur granules are generally believed to occur only in cells of the lymphocytic types—and then in more mature cells—and often only with special stains. Pappenheim calls these bodies *myeloid* azurophilic granules. Naegeli, however, vigorously disputes this interpretation and classifies the granules as unripe neutrophilic.

As to the red cells, there is little to be said. Normoblasts are present in every preparation, diminishing in number toward the last. A moderate anisocytosis, with a tendency toward the small cell, is present. There is also slight poikilocytosis.

The blood platelets are considerably diminished.

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SITUS VISCERUM INVERSUS TOTALIS

By FREDERICK TICE

Chicago

Transposition of the internal organs, either complete or partial, has long since ceased to be a curiosity, but it still maintains much interest for the embryologist, the pathologist, and more especially for the surgeon and internist.

Petrus Servius, 1643, is given the credit of recording the first case. Grueber, in 1863, was able to collect 78 cases, while A. Pic, up to 1895, collected a sum total of 195. Pollack and Jewell, in 1910, found that during the preceding fifteen years 128 articles on this condition were published, and from which they were able to analyze 78 cases. Since 1910 up to the present year 161 additional cases have appeared in the literature.

At first the condition was most frequently determined in the autopsy or dissecting room; later, by the surgeon or internist, while the marked increase during recent years is to be attributed to more accurate clinical methods and especially to the assistance of the Röntgen rays.

During the past few years four cases with complete transposition have come under my observation. Two of these were reported some time ago, but will be included, as more satisfactory skiagrams have since been obtained.

Case 1. The following abstract is taken from the original report:

(a) Clinical History: W. C., male child, eight years old. Has always been well until about two months ago, when he complained of headache, slight chills, nose bleed and loss of appetite, and his mother thought he had a fever. After a few days, as his condition did not improve, he was placed in bed and was first seen at this time. Suspecting, from the history, a possible typhoid, the abdomen was examined. Several typical rose spots were present, and the left hypochondrium examined for the confirmatory palpable spleen, which could not be detected. It was at this time, before the systematic routine examination, that my attention was directed to the right-sided portion of the heart.

Thinking that other organs as well as the heart might be misplaced led to the discovery that the liver was on the left, and the spleen on the right side. At the time of the first examination, during the typhoid, the spleen was easily palpable two fingers below the right costal arch. After a mild course of about three weeks' duration, patient made a complete recovery.

(b) Physical Examination: General physical condition is good. Head and neck are negative.

Chest.—(1) Heart: Inspection and palpation reveal the apex beat in the right fifth intercostal space about one inch inside of the

nipple line. Cardiac outline by percussion shows the base to be at the upper border of the third right costal cartilage and rib, extending about one inch to the right of the sternum. The left border is at the left border of the sternum. The right border from the right end of the base line to apex beat. Auscultation is negative, except that maximum intensity of tones corroborate location of apex as previously indicated. (2) Lungs: Negative, except normal pulmonary signs are present where cardiac findings should be and such are present to the right of the sternum. Traube's space is absent on the left, but present on the right.

Abdomen.—(1) Liver: Percussion determines liver dullness, which is located on the left side, with gastric tympany on the right. (2) Spleen: Dullness on the right side—not palpable.

Extremities.—Patient is right-handed, but uses left almost as much.

Genitourinary Organs.—Right testicle is more dependent than left. To assist in the more accurate location of the heart, liver and spleen, a röntgenogram was made.

Believing that the stomach is also transposed, the patient was given one ounce of bismuth subnitrate and a second röntgenogram was made. The most interesting feature consists in the possible mistaken conclusions to which the condition might lead. Some of these are the following:

1. With an obliteration of Traube's space, dullness in the lower left chest and the heart displaced to the right, the diagnosis of a left-sided encysted pleurisy with an effusion is quite possible.
2. Dextrocardia, congenital or acquired, could be diagnosed if the other conditions were not determined.
3. The impossibility of palpating the spleen in the usual location in those conditions in which it is enlarged, might cast doubt on the probable diagnosis as first occurred in this case.
4. In a case of cholelithiasis, the pain would be located on the left side. This condition, associated with jaundice and absence of the hepatic dullness in the normal location, might indicate the existence of an acute yellow atrophy of the liver, which diagnosis was actually made in one recorded case.
5. As other organs are transposed, it is reasonable to conclude



CASE I. FIG. 1

Skiergram of the chest and upper abdomen. Patient lying on chest. Heart and spleen to the right; liver to the left.



CASE I. FIG. 2

Stomach shadow after bismuth meal. Patient lying on abdomen. Stomach to the right.

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CASE I. FIG. 3

Colon, cecum and lower bowel shadow.
Cecum and appendix to the left.

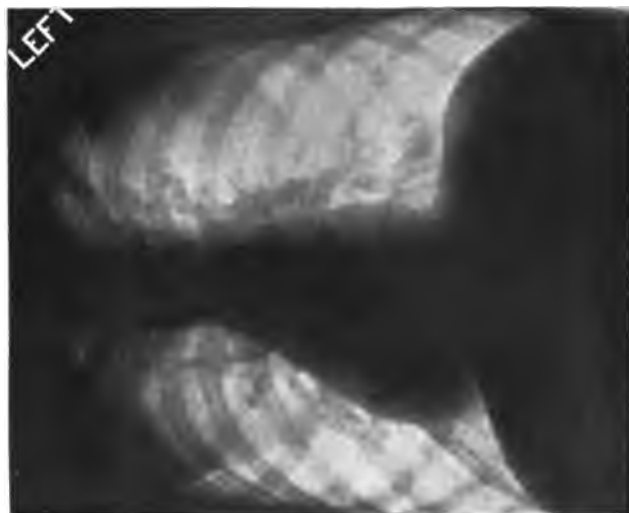
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CASE II. FIG. 4

Skigram of chest. Heart to the right.



CASE III. FIG. 5

Skiagram of chest. Heart to the right.

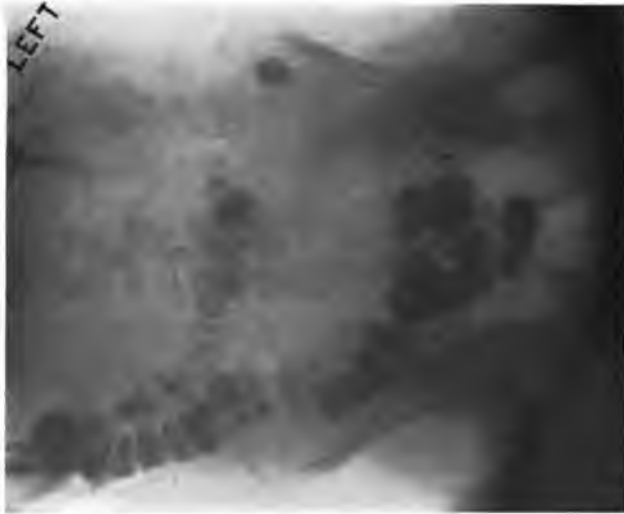


CASE III. FIG. 6

Stomach shadow after bismuth meal. Patient lying on back. Stomach to the right.

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CASE III. FIG. 7

Ascending and transverse colon after rectal injection
of bismuth. Patient on back.



CASE IV. FIG. 8

Skiagram of chest. Heart to the right.

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that the appendix is on the opposite side. This being true, in a case of appendicitis the findings would be on the left.

Case 2. Elva J. Otis, female, fifty years old. Admitted to the County Hospital, Chicago, August 18, 1911.

Present Complaint.—Paroxysmal attacks of severe stabbing pain, coming on shortly after eating. Between paroxysms pain is constantly present, but of dull, aching character. Pain begins in epigastrium, radiates to left costal border, but is especially referred to left shoulder and back. With paroxysms patient vomits large quantity of greenish material. Vomitus has never contained blood.

Past History.—Peritonitis about twenty-five years ago. Operated and drained for many months. Ventral hernia occurred, was operated and resulted in fecal fistula, which was operated on and followed by return of ventral hernia. Has had pneumonia and pleurisy, but does not know which side was involved.

Personal History.—Uses alcohol in moderation. Denies specific infections. For past twenty-five years has used large quantities of morphin.

Physical Examination.—Chest: Lungs negative, except for bronchitis; normal resonance in cardiac area to left of sternum. Traube's space dull. Cardiac dulness to right of sternum with apex beat in fifth right interspace, about two inches from the right sternal border. Soft systolic murmur at apex.

Abdomen.—Ventral Hernia: Hepatic dulness to left of median line and lower border palpable about one inch below costal arch. Spleen not palpable.

Patient was discharged September 2, 1911, on the clinical diagnosis of morphinism and transposition of viscera. This patient was again admitted to the hospital December 10, 1911, with practically the same history and physical findings, except she was deeply jaundiced. Receiving-room diagnosis was cholelithiasis and splenomegaly. Patient was operated December 15. Median incision was made. Abdominal organs completely transposed. Liver enlarged and cirrhotic. Gall-bladder much distended, but contained no stones. One large calculus impacted in common duct, which was incised and stone removed. Gall-bladder was also drained. Patient died about twenty-three hours after operation.

Autopsy Report.—Confirmed clinical findings of complete transposition of internal organs.

Case 3. Chas. Schuppel, male, forty-three years old.

This case was reported by Dr. Chadbourne and probably by several others since that time. During the past several years he has been admitted to the County Hospital every few months on various complaints. Once he had a traumatic injury to his left hip, while on another occasion he presented the symptoms of appendicitis in the left lower quadrant of abdomen. He practically lives in one hospital or another and goes from clinic to clinic exhibiting himself for a compensation. He is a typical "dispensary floater" or "medical freak." His skiagrams will only be given as a supplement to the previous report.

Case 4. P. Schoenbrun, male, thirty-four years old. (Referred by Dr. F. Chauvet, April, 1915.)

Patient was always well until about one month ago, when he suffered from slight pains and discomfort in the right lower chest. The first physician consulted informed him that he had a pleurisy. Later another physician detached the heart to the right and found the liver and spleen transposed. Fluoroscopic examination confirmed the physical findings and also no evidence of a pleurisy. It is more than probable that the discomfort in the right side was due to some cardiac disturbance from overindulgence in coffee or tobacco. A skiagram of the chest was made and arrangements completed for gastrointestinal ones, but the patient has failed to return.

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CLINICAL STUDY OF A CASE OF EPILEPSY APPARENTLY OF INTESTINAL ORIGIN

By EDWARD E. CORNWALL

Attending Physician, Williamsburg and Norwegian Hospitals; Consulting Physician, Bethany Deaconess Hospital
 Brooklyn—New York

The patient whose case is described in this paper was referred to the writer by Dr. A. C. Brush. He is a man twenty-one years old. No member of either his father's or mother's families was ever known to have had epilepsy, and his family history, as far as could be ascertained, was generally good. Concerning his personal history previous to the beginning of the present illness, the following facts were learned: He had cholera infantum in his second summer, whooping cough at five, measles at fifteen, and influenza at eighteen. Between fifteen and eighteen he grew very rapidly, increasing eight inches in height and forty-four pounds in weight during that period. He suffered from constipation as far back as his memory carries, and as far back as 1907 the constipation was so severe that he would sometimes go without a

movement of the bowels for as long as a week. He also suffered from headaches. At eighteen he entered Columbia University in the electrical engineering course. He stood well in his classes, but took no interest in athletics or outside amusements, and spent most of his spare time in the study of wireless telegraphy. The only formal exercises which he took were those in the gymnasium work required by the university. In connection with this required gymnasium work, he noticed that running often produced a sharp pain in the right upper quadrant of his abdomen, or a sensation as if he had "a heavy weight or lump there, which bumped up and down, especially down." His general health he considered to be good until the present illness began.

January 7, 1913, he ate boiled cabbage at his evening meal, and later in the evening fruit cake. During the night following he was distressed by a pain in the pit of his stomach, and in the morning, when he attempted to get up, felt so uncomfortable that he was constrained to remain in bed. About an hour after his attempt to get up, he was found unconscious, with set eyes, breathing stertorously. Twenty minutes later he revived, got rid of considerable gas from his stomach, and was relieved of his abdominal distress. This attack was diagnosticated as acute indigestion.

February 13, 1913, his college examinations took place, about which he worried, although he passed them creditably. At dinner that night he ate creamed cabbage. The following morning he awoke without special bad feelings, so he said afterward, but at 9 A.M. he was found unconscious, with saliva dribbling from his mouth, and a purplish face. Later small red spots appeared on his cheeks immediately under the eyelids, which faded out in a day or two. After this attack he was forbidden to return to college by his attending physician, and was put on a diet in which starch was restricted.

In March, 1913, he had an attack of scarlatina of moderate severity, from which he made a good recovery.

July 26, 1913, he went as wireless operator on a steamship sailing to Panama. On this trip he was much troubled with indigestion and constipation.

August 14, 1913, he returned from Panama, and immediately on arriving at his home lay down for a nap. A little later he was

found unconscious, with hands and feet moving convulsively. After inhalation of ammonia from a strong solution, he promptly revived.

September 28, 1913, at about 8 A.M., he was heard to breathe stertorously, and was found unconscious, with eyes set and tongue bitten. He revived promptly after inhalation of the ammonia solution.

October 26, 1913, after visiting away from home and eating strange food, including "chicken" salad probably made from veal, he was observed, at 6 A.M., while still apparently asleep, to be moving his legs in a convulsive manner and to be dribbling bloody saliva. He could not be aroused at first, but ten minutes after inhaling ammonia smelling salts he recovered consciousness and belched gas from his stomach; his right eye was blood shot.

Attacks similar to those above described occurred on the following dates: November 27, 1913, January 9, January 27, January 31, February 24, March 3, March 18, April 2, April 23, May 10, May 23, and May 28, 1914.

At the time of these attacks and for short periods before and after, he suffered from coated tongue, bad taste in the mouth, foul breath, belching of gas from the stomach, intestinal flatulence, giving off of offensive gases from the bowels (which had a "chemical laboratory smell") and constipation. He also suffered from these symptoms, though in less degree, off and on between the attacks. He usually had no remembrance of events occurring during the first half-hour after the attacks, and for a day or two after the attacks his recollection of events which occurred during the day or two preceding them was imperfect. He was usually drowsy for a short time after the attacks, but less so after the later than the earlier ones. It was noticed in the period between the middle of April and the end of May, 1914, that he often moaned in his sleep and twitched with his hands and feet.

May 30, 1914, two days after the occurrence of the attack last mentioned, he first came under the writer's observation. Physical examination made on that date showed: General appearance good; height, 5 feet 11½ inches; weight, 154 pounds; lungs, negative; heart apparently normal in size, no murmurs heard, action slightly irregular; area of liver dulness slightly increased; spleen, negative;



FIG. 1

Seven hours after bismuth meal. Shows bismuth collected in terminal portion of ileum (which is dilated and lies adjacent to cecum and pelvic wall) and ascending colon.



FIG. 2

Twelve hours after bismuth meal. Shows bismuth entirely in colon.

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FIG. 3

Twenty hours after bismuth meal. Shows bismuth in colon as far as rectum, where it is massed.



FIG. 4

After injection of bismuth emulsion into colon per rectum. Shows entire colon, insufficient ileocecal valve, and terminal portion of ileum, which extends downward (in all the pictures) and seems to lie or be attached to the tip of the rectum.

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transverse colon seems to dip down on the right side, cecum and ascending colon seem moderately dilated; blood pressure, 115 mm. Hg. systolic, 55 mm. Hg. diastolic. Examination of urine, June 12, 1914, showed: Quantity in 24 hours, 1914 c.c.; acid; specific gravity, 1.016; no albumin, glucose or casts found; indican, a trace; urea, 1.2 per cent.; many crystals of triple phosphates in sediment.

The plan of treatment laid out for this patient was as follows:

He was kept out of college until February 3, 1915, but allowed to amuse himself with the wireless telegraphic outfit which he had rigged up on his house, and encouraged to take exercise in the open air.

He was put on a non-putrefactive, laxative diet, arranged to supply daily about 75 grams of protein and fuel of the value of about 2,500 calories. The articles in his dietetic prescription were: Milk and preparations of milk, including lactacidized milk, cream cheese, cottage cheese, American cheese, cream and butter; cereal preparations, including bread, rusk, zwieback, maccaroni, boiled rice, bran biscuits and raw wheat bran; potato, carrot, and specified green vegetables, the latter in good quantity; specified fruits, of the kinds containing citric and malic acids, in good quantity; milk soups made with milk and specified vegetables; olive oil; levulose.

Certain exercises were prescribed for him, to be performed before going to bed. These exercises were given for the purpose of improving peristalsis, strengthening the abdominal muscles, and restoring as far as possible to their original position his displaced viscera. They included the exercises known by the names of "pumping," "rotation," "scissors," and "bicycle."

He was given cathartics of the vegetable class as needed, and also Russian oil.

He was given rhubarb and soda mixture in dram doses three times a day, and sodium bromid in ten-grain doses three times a day.

This plan of treatment, as regards the diet and exercises, has been steadily kept up to the present time, June 7, 1915. The sodium bromid was discontinued after one month. The Russian oil was discontinued after about three months, as the patient did

not think it had much effect on his bowel movements. The rhubarb and soda mixture was taken intermittently. The cathartics were kept up, in varying doses, until the end of May, 1915, when it was found that the bowels moved satisfactorily without them, while raw bran was included in the diet.

Seven days after beginning this treatment, that is, on June 7, 1914, the patient had a convulsive attack similar to those previously described, except that the symptoms were less pronounced and the tongue was not bitten.

It was not until nearly six and a half months later that another attack occurred, which followed the patient's first attempt to smoke tobacco (as a celebration of his coming of age). At 1 A.M. December 18, 1914, shortly after going to bed, he had a slight attack, which lasted less than two minutes, in which his tongue was not bitten, and from which he recovered promptly and completely. There was no loss of memory of things that happened shortly before the attack. About half an hour after recovering he had a regular movement of the bowels.

He resumed his interrupted course at Columbia University on February 3, 1915, returning every night to his home, which was in Flatbush. He was not allowed to take part in the regular gymnastic exercises required by the college.

February 24, 1915, at 6.30 P.M., while sitting at a table writing, he suddenly fell off his chair, unconscious, but did not bite his tongue. He recovered in about one minute, apparently completely. Just before the attack he remembers that he started to write the letter C, but he wrote the letter L instead. About fifteen minutes after recovering he answered a call on the telephone, and spoke rationally, but he was not able later to recollect what the conversation was about, although he remembers clearly something which happened about three minutes after the attack; and he appeared perfectly normal to those who observed him immediately after the attack. That night red spots, a little larger than a pin head, appeared on his arms and legs, around his left eye and over the left end of his upper lip, which were faint the following morning and disappeared before the next night. Immediately after this attack and during the entire evening, he had a feeling in his head "like what one has in a stuffy room."

After returning to college he did not get as much sleep as formerly, averaging only about six and a half hours daily.

April 12, 1915, he had an attack similar to the last one described, but no red spots appeared. This attack occurred while sitting at a table studying. Immediately after coming out of it he talked incoherently about his studies for a few minutes. He recovered completely in less than half an hour. There were no premonitory symptoms. On the day of this attack he had been irritated and excited by a controversy with one of his instructors, and also had a slight coryza.

May 11, 1915, he had another light attack similar to the last three in general character, being of short duration, and not attended with biting of the tongue. This attack, like the last two, occurred while he was sitting at a table studying.

In the latter part of May, 1915, he passed his college examinations with credit.

Physical examination on May 23, 1915, showed him to be in good general condition. Weight, 159½ pounds, which was 5½ pounds more than when he began the present treatment. Pressure over the ascending colon produced a sensation "as if something were drawn tightly over it," especially over the upper half.

X-ray examinations were made of this case by Dr. Charles Eastmond on June 1, 2 and 3, 1915. His report is as follows:

"Seven hours after a bismuth meal:

"The stomach is entirely empty. There is a large residue in the terminal ileum, which is considerably dilated as a whole, but there are no local points of constriction, nor is the dilatation greater in one part than in another. The bismuth has progressed to the hepatic flexure.

"After twelve hours:

"The ileum is entirely empty. The bismuth is distributed throughout the ascending, transverse and descending colons. The first portion of the transverse colon lies in contact with the ascending colon, but the incisures are sharp. The colon at this time presents no other abnormalities, except that the transverse portion is excessively long, so that even in the prone position it descends into the pelvis. With the patient standing at this time the hepatic flexure is at a point about one and a half inches above the iliac

crest, and the splenic flexure only about three and a half or four inches above the iliac crest. The transverse colon in this position lies well down in the pelvis.

"After about thirty hours:

"The cecum, ascending colon and first half of the transverse colon have emptied fairly well. The bismuth now fills the colon right down to the rectum. The transverse colon still appears to be very long and descends well down into the pelvis.

"The patient was ordered a cathartic and asked to return.

"After a bismuth enema had been administered:

"The enema has passed the ileo-cecal valve so that the last portion of the ileum can be distinctly seen; it comes up from below the tip of the cecum, against which it seems to rest, and lies mostly against the cecum until it enters it. The entire colon is distinctly outlined, and in both the prone and erect positions the colon presents practically no abnormalities except for the excessively long transverse colon mentioned previously and the abnormal descensus of the colon into the pelvis."

Of the facts observed or elicited in the study of this case, the following, perhaps, are among the more significant:

1. Facts in the family and personal history previous to the onset of the convulsive seizures:

The family history was negative. In the patient's previous personal history constipation appears prominently, with gastric and intestinal flatulence and discharge of foul gases from the bowel. There was a peculiar sensation experienced in the upper right quadrant of the abdomen, brought on by running, described as a "feeling as of a lump bumping up and down," which was sometimes accompanied by pain. For some months before the convulsive attacks appeared, the patient devoted himself closely to study and neglected ordinary amusements, social diversions and out-of-door exercise.

2. Physical findings:

The most significant physical findings are those revealed by the X-ray examinations, which showed a coloptosis, with the transverse colon in the pelvis; the ascending colon extending one and a half inches above the iliac crest, and the descending colon extending three or four inches above the iliac crest; notable dilata-

tion of the terminal portion of the ileum, and insufficiency of the ilio-cecal valve; but no distinct obstruction anywhere in the course of the gut, and no particular stasis except in the terminal portion of the ileum.

3. Facts observed in connection with the bowels:

The constipation showed a tendency to persist unless relieved by laxatives. Russian oil did not prove of much value. Moderate doses of laxative drugs of a vegetable character, in connection with a diet which included cellulose and other organic laxative substances, were sufficient to produce satisfactory movements once or twice a day; and at the end of the year the diet alone was sufficient. Coated tongue, bad taste in the mouth, foul breath, belching of gas from the stomach, discharge of offensive gases from the bowel were common symptoms during the period before coming under the writer's observation in which the attacks occurred, and were particularly prominent shortly before and after the incidence of the attacks; but they were much less prominent, and, indeed, almost unnoticeable during the period while under the writer's observation and treatment. During this last period discharge of gases from the intestines was occasionally noticed, but the gases no longer had the "chemical laboratory smell," but had an odor like that "from a flatulent horse." The stools during this last period, while on the diet prescribed by the writer, were notably smaller in size than formerly.

4. The convulsive seizures:

The convulsive seizures resembled, in general, those commonly observed in epilepsy, but they showed considerable variation in character, intensity, duration, and the occurrence of premonitory and subsequent symptoms. They occurred six times during the year 1913, and eleven times between January 1, 1914, and June 1, 1914. Their rate of occurrence was about once a month during the first year, except for five months following an attack of scarlet fever; and a little more than twice a month during the first five months of the second year, at the end of which period the patient came first under the writer's observation. After being put on a course of treatment which was aimed chiefly at relieving constipation and intestinal putrefaction, the attacks diminished greatly in frequency. One attack occurred seven days after beginning the

treatment, and then there was freedom from the attacks for more than six months. During the remainder of the period of a little over a year, while under this treatment, four attacks occurred, which were milder and less typically epileptic in character than the former ones. Three of these four attacks occurred while the patient was occupied with his college work, and was getting an insufficient amount of sleep; and they took place while he was sitting at a table studying, instead of when lying down, as before.

The study of this case, as far as it has been carried, does not yield a definite answer to the diagnostic and prognostic questions which naturally arise, viz., whether the convulsive seizures are due to an intrinsic and incurable derangement of the nervous system, or whether they are due essentially and chiefly to a cause outside the nervous system which is removable. The facts observed, however, suggest the possibility of a toxic-exciting or mechanical cause of intestinal origin, and they also suggest what seems to be the most promising line of treatment.

DIAGNOSIS FROM THE STANDPOINT OF PHYSICO-DYNAMICS

By J. MADISON TAYLOR

Associate Professor of Non-Pharmaceutic Therapeutics, Medical
Department, Temple University
Philadelphia

I

Any clinical problem deserves to be approached from at least two or three angles. Human infirmities are not only of great complexity, but greater urgency. That factor which is most urgent needs first recognition and first aid. A position of advantage being obtained by affording relief to the obvious errors, we may then profitably proceed to search out every avenue of causation and phenomenal formation.

It is not always to the patient's best welfare to select for particular treatment that one feature which, in the opinion of the clinician, seems especially interesting; nor the only one which his degree of learning or his taste or experience induces him to select

as his point of departure. Hence, the hope may be expressed that clinical teachings of the future shall include more of a perspective, more practical transvaluations of grouped phenomena, also their correlation and just apportionment of blame. In the survey certain significant facts demand inclusion which are too often subordinated.

Any one of us can recall instances where we happened to note clearly the one point constituting a key to the situation which had escaped the attention of many able predecessors.

The personal equation, we say. Yes, it is inevitable that some consciousnesses are able to perceive one kind, others yet another kind or form of origin or determinant of action. The fact is if we can learn to simplify our means of approach by new and promising directions, they are worth considering.

II

A sick human being is something other than a living body afflicted with a damaged organ, or disease processes or effects. To relieve the malady, it is seldom enough to define just what, where and how the major phenomena are and are manifested, and to administer suitable counsel or medication.

The mind, always a dominant factor, is contained in, and conditioned by, an essentially mechano-physical organism.

The sick body is an aggregation of biochemical forces which have become perverted and require particularized regulation. It is also a human mechanism out of gear.

A multitude of physico-dynamic factors likewise co-exist demanding recognition and adjustment. The term physico-dynamics seems a good one to describe that department of bio-dynamics which has to do with physics, the laws of matter and energy.¹

The laws of physical science are universal and apply equally to

¹The term physics is a poor one, so is physical or physiologic, since all are based on physics or growth forces. Hence, it is by no means easy to get a word which will convey the idea of what is meant when we wish to differentiate between the forces or phenomena described in the science of physics as contrasted with the more vitalized processes of active growth energies or physiology. It is worse when we would attempt a concept of physical phenomena. In despair of better orthography, the term physico-dynamic has been selected to meet the need of the forces which are to be connoted as residing in the science of physics.

living organisms and to so-called inanimate objects. Maladies should always be estimated from the effects they produce on (1) the mechanisms of the individual, and (2) his or her personal peculiarities, which differ in kind and degree from others, as well as from causes and abnormal effects common to others.

Any clinician would be wise to read over, once in a while, an elementary book on physics. The mind will thus become re-familiarized with properties of matter, energy, motion, velocity, gravity, statics, etc., helpful in understanding biologic principles.¹

Lord Macaulay urged every one to read Euclid once a month to aid in keeping the mental processes systematic and sequential. We might with equal emphasis recommend reading over a primer on physics.²

Physics deals with molecular changes of matter. Mechanics deals with the laws of energy and motion. Psychology deals with the facts of consciousness; with classification and generalization of mental phenomena. Geometry deals with spatial facts. Chemistry deals with atomic combinations and mutations; different forms of matter. Physiology deals with processes going to make the equilibrium of organic life. Sociology deals with processes of social life, etc. Biology deals with the properties of matter and energy of living matter. The lines of demarkation between them are not defined; they interact.

The era of "expectant treatment" is not so large a credit to medical progress as at first we were led to believe. To be sure, inherent force, making for growth and repair, can be relied upon to a certain point; then skill, experience, shrewd observation and common sense must fill in the outline. This outline must have as

¹Dr. John C. Draper's book on "Medical Physics" (Lea Bros., 1885) is a mine of information.

²Two courses of action should arise in the adviser's consciousness in times of need. One is to act promptly guided by inferential interpretations, and to supply such imperative help needed as previous experience suggests, a hit-or-miss, rule of thumb, rough and ready method, e.g., to fix a watch we may listen to it, note disorders in action, may proceed to oil it, move its hands, shake it, and perhaps it will go. The second and more certain way is to secure precise information as to abnormal conditions and remedy them. In the matter of the watch, e.g., to open it, and, after carefully inspecting its mechanism, to repair precisely what is found to be amiss.

a basis certain scientific factors and principles as are involved in the problem.

III

In making diagnoses from the physico-dynamic standpoint, it is desirable to acquire a somewhat different method of securing, as well as interpreting, findings; to use the familiar forms of inspection, palpation, etc., but to look and feel for other conditions than those commonly sought for. For example, a multitude of abnormalities will be revealed by expert palpation, tactile awareness, resistance or relaxation, densities, spasm, vague sensitiveness, altered local static relationships, etc., etc.

Among the factors to be reckoned with are:

1. Anomalies of development, especially minor departures from the norm. These by faulty habitudes, disease processes, errors in conduct, accident and the like fortuitous happenings, may become emphasized or exaggerated and impair organic competence.

2. Anomalies of structure induced by neglect of right methods of living, especially those bearing on faulty mechanics, errors in action, movement, posture, inducing disorders of mobility, elasticity, pliancy, tone, rhythm; also direct and by effects of over-effort, exhaustion, of disuse or misuse by neglect of normal exercise or excitation.

3. Abnormalities of structure arising from disordered or diseased states, and of traumata, etc.; (a) static effects of errors in metabolism, minor infectious processes, injury, upon gross structures and finer cell aggregations, also on regulative mechanisms, ductless glands, sense organs, nerves, brain, and the like; (b) resistance effects, spasm, rigidities, densities; (c) sensory effects. Over-excitation on sensori motor mechanisms, negative anesthesia or positive hyperesthesia, pain direct or indirect, immediate or remote or reflex complicated effects.

4. Reflexes, their phenomena and effects in solving clinical problems; reflexology, reflexodiagnosis; referred pains, origin and manifestation; aberrancies of reflex phenomena.

IV

In employing physico-dynamic diagnosis, it is desirable to become particularly adept in familiar forms of investigation, which

are ordinarily used perfunctorily, or only to reveal a limited group of findings.

Inspection: One should be so familiar with normal gross anatomy, posture, attitude, proportion, etc., as to observe at a glance departures from norm, anomalies in conformation and to appraise causative factors, among which are effects of local relaxation, spasm, contracture, rigidity, displacement, imbalance and the like mechanistic phenomena; also to become aware of normal or admissible variants.

Palpation: Using this term in its widest, most liberal interpretation of tactile perception or apperception, the "feel" of structures, superficial and deep; estimating degrees and kinds of tissue-tension, spasm or relaxation, capabilities of action, mobility passive and active; of adhesions, densities, rigidities; of resistances or counteractions, also relaxations; of sensitiveness, normal and abnormal; of dryness or moistness; of local temperature changes, and the like; above all, an instinctive quality not definable, but rather an *awareness* of something amiss in the substance or fabric, and what it probably indicates to be learned through tactile perceptions.

Diagnostic Manipulation or Handling: While seizing and testing tissues or parts by larger motions than touch on surfaces may be regarded as part of "Palpation," I submit that it is worth separate and careful consideration. By this imitation of, and indeed improvement on, the methods of the surgeon, a different group of phenomena are evaluated than the relatively crude seizings and fingerings they ordinarily employ. The internist can thereby determine the significance of a multitude of phenomena entirely within his domain.

Some observations thus made are exact and some safely inferential. To be sure, the internist does make use of some, a few, similar explorations, but by no means habitually, nor does he learn as much thereby as he might to advantage. It is this diagnostic measure I would direct attention and hope to show its value.

For example, much will be revealed by imitating the surgeon by seizing the limbs, moving them about, determining degrees and directions of resistance, limitations of mobility, pliancy caused by

various agencies among which are effects of metabolic disorders, gout, rheumatism, fibromyositis, etc.

Especially is this of use in searching for latent forms and degrees of that widely prevalent and, to my mind, unappreciated group of disorders known as fibromyositis. These are sometimes painful, latently tender, at others non-sensitive or only sensitive occasionally, or only by cunning palpation, and hence particularized definition, yet are at all times more or less disabling.

V

Conditions revealed by these three avenues of approach will be found often of large significance as bearing on circulation not alone of the major, but especially of minor (lymphatic) cycle. As to the major circulation, so large a subject, we can here merely allude to some points which need special emphasis in this connection, e.g., vagus tone, sympathetic, vasomotor tone, and tone in the autonomic distribution.

In particular, attention should be focussed on lymphatic permeability, or stasis, and its bearing on nutrition of vital organs, nerves, nerve-sheaths, muscles and muscle-sheaths, attachments, fibrous structures, and the like.

The disorder presenting is to be estimated from observed effects, now existing in the individual, in the light of personal peculiarities (inherent or acquired), which may differ in kind and degree from others, as well as from causes differing in effects on others.

In short, a study is to be made of what kind of human mechanism is affected with the disorder; how he or she reacts to the mechanistic group of causal factors; rather than the more usual method of merely determining the clinical phenomena presenting, and applying remedies presumably capable of modifying the condition found.

Moreover, there are vague, unclear, morbid, often painful, states to be reckoned with in many diseases which complicate the plainer manifestations, but are too often regarded as inevitable and bound to disappear as the disease processes yield to natural powers for repair.

It is my purpose to present reasons for believing that many

of these contributory factors can and should be discovered, defined and delimited, with the result of modifying the distressing or ultimately damaging effects otherwise ignored.

VI

Physiologists have given us a mass of information which it is our duty to interpret and use. Among the items of available knowledge of which clinicians do not make adequate use is *physiologic physics*.¹

The laws of physical science are universal and apply equally to living organisms and so-called inanimate things.

Clinicians have contributed much to this department, notably since the advent of the Röntgen ray.

Surgeons, especially by laying bare secrets of bodily makeup, are enabled to see, touch, and thus know a vast amount of mechanistic facts found in normal and abnormal states, as well as changes wrought by disease. These opportunities are becoming utilized increasingly by internists, and the time will soon come when the findings will be presented in systematized form to the great advantage of the general practitioner, and especially to the patient.

One element of common sense consists in determining the nature, peculiarities and abnormalities of the individual mechanism; whether, and if so where, it has undergone changes in shape, tone, balance; whether some parts are too loose or too tense; whether the fountains of force are rightly supplied, transmitted, transformed, or interfered with; too rigid or too mobile or in other respects out of adjustment.

When so complex a mechanism as the human body is out of gear in one particular, it is a truism that other parts share in the disharmony, and that less obvious derangements become efficient factors in distress, disability, or even grave peril. When the or-

¹The *Iatro* physicists of the seventeenth century (in Italy), as Abrams remarks, were enthusiasts in a most important domain of medicine. They sought to explain the functions of the body, and the effects of remedial agencies through static and hydraulic laws. Though long neglected, these principles must be reckoned with to-day, or our duty to an invalid is not fully discharged. Some legendary notions still obtain exhibiting the primitive hope for "specifics," for "panaceas," and other mythogenic expectations of effecting cures.

ganism is thus in disequilibrium there is need for a master workman to readjust and redirect autoprotective forces.

Röntgenotherapy and surgery have brought us a long step forward in knowledge of function and of obscure disorders, especially of mechanistic or static derangements. Surgery has shed much light by direct touch and sight, where heretofore only inferences could be made.

These new and practical opportunities of learning things as they actually are have already revolutionized observation, carried knowledge to a point unbelievable a few years back. Not yet have internists availed themselves of these priceless visualizations direct and mental, as they soon will do. The most thorough laboratory (biochemical) researches can only reveal abnormalities within a limited domain.

VII

Admitting all that these liberalizing sources of awareness can supply, there yet remains a number of significant collateral conditions requiring elucidation. Any one who will take the trouble to survey the abundant sources of diagnosis by expert observation, by vision, touch, handling of structures, and at the same time holding in mind his primary knowledge of physics, will greatly increase his powers and usefulness for relief and cure.

Doubtless most, if not all, who study medicine have been grounded in the principles of physics, the essential properties of matter, and can call them to mind. The special applicability of these forces has been demonstrated in teachings of biology, in physiology and anatomy.

To judge from personal experience in teaching students of medicine, both under- and post-graduates, the impressions received from these studies fluctuate and often become practically effaced. To cite my own case, an exceptionally good grounding in physics had faded sadly when occasions arose to utilize my knowledge of physics in forming clear concepts of primitive or essential forces as manifested in physiologic and anatomic problems. It is fair to assume few keep them clearly in the foreground of consciousness during the solution of daily recurring fields of inquiry.

Progress of Diagnosis and Prognosis

GENERAL METHODS OF EXAMINATION—SYSTEMIC AFFECTIONS—DISORDERS OF GENERAL METABOLISM

Toxicity of Urine—M. H. ROGER, *Presse méd.*, May 31, 1915.

In his analyses of urine Bouchard succeeded in dissociating the effect of seven different substances, to which are added four discovered later. We know at present: a diuretic substance, urea; a narcotic substance, a sialogenous substance, two substances inducing convulsions, a myotic substance, a hypothermic and a hyperthermic substance, a cardiac poison; a hypotensive and a hypertensive substance, these latter discovered by Abelous and Bardier. Urine emitted by day is narcotic; that emitted by night is convulsivant, according to Bouchard, which leads him to maintain the toxic effect of sleep.

ZIMMER.

Symptoms of Urinod Poisoning—F. A. HARTMAN, *Arch. Int. Med.*, July, 1915.

Urinod is prepared from the distillates of acid-treated urines. It is a neutral malodorous oil, boiling at 108 degrees C., with 28 mm. pressure. It is a cyclic ketone with the empirical formula C_6H_8O . The symptoms produced by urinod are nausea, headache, loss of appetite, heaviness of the stomach after eating, twitching, irritability, mental dulness, physical weariness, drowsiness, dyspnea, convulsions and a state of nonirritability. Urinod appears to be one of the most toxic substances in the urine. Cases are cited in which there have been indications of urinod retention in the body. The symptoms of urinod poisoning resemble the nervous symptoms of uremia. Urinod retention, therefore, might partly account for these nervous symptoms.

SACHS.

Diagnostic Value of Uric Acid Determinations in the Blood—O. FOLIN and W. DENIS, *Arch. Int. Med.*, July, 1915.

In gout the blood is almost invariably high in uric acid, while the other waste products represented in the nonprotein nitrogen of the blood are usually within normal limits. In arthritis also the blood is not infrequently abnormally high in uric acid, but most such cases have abnormally high nonprotein nitrogen as well. Neither qualitative nor quantitative determinations of the uric acid in the blood can be depended on in the differential diagnosis in doubtful cases of gout

or arthritis. The patient must be put on a purin free diet and uric acid determinations must be accompanied by determinations of the nonprotein nitrogen (or urea). SACHS.

Uric Acid Content of Infantile Blood—E. LIEFMANN, *Zeitschr. f. Kinderheilkunde*, Vol. XII, Nos. 4 and 5, 1915.

The uric acid content of the blood of the nursling fluctuates between 1.3 and 1.7 mg. in every 100 c.c. of blood. When on a diet rich in purins it increases rapidly. After withdrawal of the purins the uric acid content declines slowly. The uric acid content is also augmented in fasting, febrile and systemically diseased children. Nurslings with exudative diathesis exhibit no special increase of uric acid in the blood. For this reason, it appears, that there exists no connection between the exudative and the uric acid diathesis.

MILL.

The Cholesterin Content of the Human Blood Serum—I. H. PRIBRAM, *Zentralblatt f. innere Medizin*, No. 21, 1915.

In most cases of nephritis the cholesterin in the serum is much increased. In polycythemia this is also the case. A case of diabetic coma showed an increased amount of cholesterin in the blood serum. The cholesterin content of the serum warrants certain conclusions in the pathogenesis of various diseases.

WESTERN.

Pepsin in the Blood Serum—P. SAXL, *Wiener med. Wochenschr.*, March 8, 1915.

Author succeeded in bringing about casein digestion by the blood serum. He did this with the sera of 20 healthy or slightly diseased individuals. This casein digestion is apparently due to peptic activity.

MILL.

Blood Sugar Determinations in Diabetics and Their Clinical Import—M. LAURITZEN, *Ugeskrift f. Læger*, No. 6, 1915.

The blood sugar determinations were made according to the method of Ivar Bang. The determinations were made with the blood of 100 diabetic patients. Author believes that these determinations are of great value in the differential diagnosis. (1) Whether the case is one of diabetes with or without hyperglycemia; (2) in the forming of a prognosis in the cases in which acetonuria and diaceturia permit of no orientation as regards an unfavorable prognosis, and (3) to control the results of the therapeutic endeavors. While, formerly, the aglycosuria seemed sufficient proof of the success of the instituted treatment, one can now determine the diet which does not give rise to hyperglycemia after the meals. Such a diet is to be kept by the diabetic patient as long as it is possible. Hyperglycemia, it must be known, destroys the tolerance for starches in diabetes.

TESSEN.

Permanganate Test for Spinal Fluid—E. LACKNER and A. LEVINSON, Arch. Pediat., July, 1915.

The test is a modification of the Kubel-Thiemann method, but in the following form was first employed by Mayerhofer. The method is as follows: One c.c. of the spinal fluid is measured in an accurately graduated pipet and introduced into an Erlenmeyer flask; 50 c.c. distilled water and 10 c.c. of diluted H_2SO_4 are added and the mixture is brought to a boil; 10 c.c. of a decinormal permanganate solution is then introduced into the flask and the solution is boiled for exactly 10 minutes. At the end of the time 10 c.c. of decinormal oxalic acid is put into the flask, whereupon the red or yellowish-red color turns white. Titration is carried on drop by drop from a buret containing permanganate solution until the color of the solution in the receptacle turns red and remains so for a number of minutes. The number of c.c. of permanganate required to produce the end reaction is then read off and the figure is taken as the permanganate index. In doing this one must, however, make certain that 10 c.c. of N/10 permanganate equals 10 c.c. N/10 oxalic acid. It is also necessary to ascertain how much permanganate is required to oxidize the water and the H_2SO_4 , and this amount should be subtracted from the number of c.c. of permanganate required to oxidize the spinal fluid solution. For example, if 4 c.c. of N/10 permanganate was required for the spinal fluid and 0.5 c.c. permanganate for water and H_2SO_4 , the 0.5 c.c. should be subtracted from the whole number, leaving the reduction index only 3.5. The spinal fluid in normal cases, or even in meningismus has little organic substance. As a result the permanganate index is low, while the organic substances in the spinal fluid of meningitis are increased, and therefore the permanganate index is higher. In tuberculous meningitis the first portion of the fluid contains more organic substances than the second, the second more than the third, each portion being about 10 c.c. in amount. In meningismus the reverse is true, the consequent portions containing a greater amount of organic substances than the preceding. These findings are hence of diagnostic value. SACHS.

Orthostatic-Lordotic and Nephritic Albuminuria—SCHOLDER and VEITH, Archiv f. Orthopädie, Mechanotherapie u. Unfallchirurgie, Vol. XII, No. 4.

This is a review of Jehle's work. The article does not bring anything new. MILL.

Theory of Basedow's Disease—A. OSWALD, Münchener med. Wochenschr., July 6, 1915.

Author is not of the opinion of Möbius, according to which the entire syndrome of Basedow's disease is primarily dependent upon a disease of the thyroid gland. He believes that an important genetic factor of the syndrome, probably the most important, is situated in the nervous system. Author does by no means deny the

participation of internal secretory activity in the genesis of the Basedow symptom-complex, and he even is of the opinion that the thyroid gland plays the most important rôle in this regard. However, he furnishes sufficient evidence that the most important of all the genetic factors is due to the state of the nervous system. The article must be read in the original. MILL.

The Thyroid of Phthisical Patients—H. KEHL, *Virchow's Archiv*, Vol. CCXVI, Nos. 1 to 3.

Anatomical examinations of the thyroid gland of individuals who had died of tuberculosis. Of 50 thyroids 2 showed tuberculosis. Lymphatic tissue was not found in any of the glands. Typical Basedow changes were also not encountered in any of the glands. Increase of connective tissue, however, was noted. MILL.

Prognosis in Exophthalmic Goitre—H. M. GERNEY, *Brit. Med. Jour.*, May 29, 1915.

From an investigation of 93 cases of exophthalmic goitre author states that the death rate is 25 per cent. in cases beginning between 10 and 15 years of age, and increases gradually to 44 per cent. in cases beginning after 45 years. In cases coming into the hospital who have been ill under one year, the death rate was 35 per cent., between 1 and 2 years 75 per cent. The rate falls after the fourth year, as after that length of time the disease may be regarded as chronic and the symptoms are always less acute. Early development of very acute symptoms, such as diarrhea and tachycardia, add to the gravity of the prognosis. SACHS.

Raynaud's Syndrome—O. T. OSBORNE, *Am. Jour. Med. Sci.*, August, 1915.

A very readable article that brings nothing new. Author emphasizes that the syndrome may appear in a very mild form; that women are attacked much more frequently than men; that the commonest age is from 15 to 30, though no age is exempt; that frequently uterine and ovarian disturbances are encountered; that the worst attacks sometimes occur periodically, with a more or less close relationship to the menstrual period; and that some of the well-known symptoms of a disturbed thyroid secretion are often present, probably more frequently on the side of hypothyroidism than hyperthyroidism. SACHS.

Hypertrophy of Thymus and Thymus Deaths—T. LE BOUTILLIER, *Arch. Pediatr.*, May, 1915.

A careful examination of the chest in the region of the thymus should be made in all children who are examined in order to determine if there be an enlargement of this gland. Especially is this so in cases of slight or marked dyspnea or cyanosis which cannot be traced to any other cause. These cases should be röntgenographed. Tracheostenosis, resulting therefrom, is a positive condi-

tion and, in certain cases, death is caused by it. Enlarged thymus is sometimes associated with goitre.

SACHS.

Hibernation and the Pituitary—H. CUSHING and E. GOETSCH, *Jour. Experiment. Med.*, July, 1915.

A train of symptoms, coupled with retardation of tissue metabolism and with inactivity of the reproductive glands, not only accompanies states of experimentally induced hypophysial deficiency, but is equally characteristic of clinical states of hypopituitarism. The most notable of these symptoms are a tendency in the chronic cases towards an unusual deposition of fat, a lowering of body temperature, slowing of the pulse and respiration, fall in blood-pressure, and often a pronounced somnolence. These symptoms bear a marked resemblance to the physiological phenomena accompanying the state of hibernation, which have hitherto been unsatisfactorily ascribed solely to extracorporeal factors; namely, a seasonal deprivation of food and low temperature. Hibernation may be ascribed to a seasonal physiological wave of pluriglandular inactivity and the essential rôle may perhaps be ascribed to the pituitary body.

SACHS.

Human Hypophysis after Castration—R. RÖSSLE, *Virchow's Archiv*, Vol. CCXVI, Nos. 1 to 3.

Castration may lead to an enlargement and the histological alteration of the anterior lobe of the hypophysis. There is found a multitude of eosinophiles, heterotopia of eosinophiles and small numbers or absence of basophile epithelia. These histologic characteristics are, however, neither constant nor absolutely specific of the hypophysis of castrates.

MILL.

Heredity of the Diabetic Constitution—I. H. PRIBRAM, *Zentralblatt f. innere Medizin*, No. 21, 1915.

Pedigree of a family the father of which is an offspring of a gouty, the mother of which comes of a diabetic family. The three oldest children have diabetes. The disease appeared in the fourth decennary of life. The younger brothers and sisters, of whom there are six, are still free from glucose. The third generation is not as yet diabetic.

WESTERN.

Ciliary Body in Health and Disease—H. P. DUNN, *Lancet*, May 29, 1915.

Author states that hypothyroidism is sometimes the cause of iridocyclitis. This form of irido-cyclitis readily responds to thyroid medication.

SACHS.

Carcinoma—J. E. ELSE, *Northwest Med.*, July, 1915.

Any epithelial cell may under certain conditions develop cancer cells. In at least a portion of the cancer group there is a contagious element and the process can be and is transmitted from one person to another. Infective organisms play a part in the etiology of some

cases, but whether they have a specific action or merely furnish the necessary chronic stimulus to cause malignant proliferation of epithelial cells, is not known. Trauma, chemical irritation and chronic irritation of all other types are of etiologic importance. WESTERN.

Skeletal Cancer or Bone Metastases—E. H. RISLEY, Boston Med. and Surg. Jour., April 22, 1915.

Metastases are more common after cancer of the breast than any other organ. The prostate and thyroid are next in frequency as a source of metastases. The liability of a bone to cancerous invasion increases with its proximity to the site of the primary focus. Thus the sternum and ribs are affected about equally and more frequently than any other bones. Pain is the only characteristic symptom. Visible or palpable tumor is rare, while spontaneous fracture is quite common. Any fracture of a long bone occurring as a result of a trivial injury should immediately suggest the possibility of bone metastases and should lead to careful search for the primary new growth. In all cases of painful paraplegia a neoplasm should be suspected.

SACHS.

Age Incidence in Sarcoma—C. V. WELLER, Arch. Int. Med., April, 1915.

Sarcoma occurs most frequently at the age period of 48 to 52. The age distribution of sarcoma in males and females is nearly the same. The sarcoma incidence in youth is somewhat higher than the carcinoma incidence, nevertheless there is throughout life a marked parallelism between the age incidence curve for the two types of malignancy, and for more than twenty there is a practicable coincidence in the age distribution.

SACHS.

INFECTIOUS DISEASES

Prognostic Value of the Temperature Curve in Pulmonary Tuberculosis

—J. SZABÓKY, Zeitschr. f. Tuberkulose, Vol. XXIII, No. 6.

The more uniform the temperature in pulmonary tuberculosis (low amplitude) the more favorable is the prognosis, provided that the low amplitude is not due to low minima alone. High amplitude is indicative of an unfavorable prognosis, even when the temperature in toto is not too high. The temperature elevation occurring in patients (that had been afebrile or became afebrile by treatment) after the performance of a certain amount of work is only of bad prognostic significance when it persists after a protracted period of rest.

FRY.

Intracellular Occurrence of Tubercle Bacilli in the Sputum—M. COHN, Beiträge z. Klinik d. Tuberkulose, Vol. XXXI, No. 1.

Examination of the freshly raised sputum in 81 cases of patients with pulmonary tuberculosis. The preparation was stained with a concentrated Assmann-Jenner stain. Author studied the behavior

of the phagocytes toward the tubercle bacilli. Among 81 cases, 8 (10 per cent.) showed marked phagocytosis. Of these 5 occurred in the 30 cases that died later on; 2 occurred in the 29 cases with a dubious prognosis, and 1 among the 11 cases with a good prognosis. The sputa with marked phagocytosis were mostly rich in bacteria. The intracellular location of the tubercle bacilli does not admit of any conclusions in regard to the course of pulmonary tuberculosis. Lymphocytes do not occur frequently in the sputum of tuberculous patients.

FRY.

Tubercle Bacilli in the Circulating Blood—C. R. AUSTRIAN and L. HAMMAN, Johns Hopkins Hospital Bull., Aug., 1915.

In pulmonary tuberculosis authors were unable to demonstrate the presence of tubercle bacilli in the blood in a single instance notwithstanding the fact that many of the patients when examined were in the last stages of the disease. In animals, when the disease remains localized in the lungs, tubercle bacilli are not found in the circulating blood. When the disease becomes generalized, as it does in the late stages of pulmonary infection and very soon after overwhelming intravenous injections, circulating tubercle bacilli are frequently demonstrable. The evidence presented points strongly against the mobilization of tubercle bacilli by the injection of tuberculin. A larger number of positive results was revealed by the method of animal inoculation than by the microscopical examination of the sediment obtained by treating blood according to the acetic-acid-antiformin method.

WESTERN.

Frequency of Tuberculosis in Childhood—B. S. VEEDER and M. R. JOHNSTON, Am. Jour. Dis. Child., June, 1915.

A study of the tuberculin tests in 1,332 hospital children in St. Louis shows that the percentage of positive reactions reaches a maximum of 44 per cent. at the age period of 10 to 14 years, including cases with clinical tuberculosis. These figures are much lower than the usual "90 per cent." figure for the incidence of infection with the tubercle bacillus in children by their fourteenth year, which has gained such widespread publicity and which is based on the figures of Hamburger of Vienna. No conclusions as to the extent of infection can be drawn from the statistics of any one city or class of children, and the statement that 90 per cent. or more of individuals are infected by puberty is an extreme exaggeration of the actual conditions which exist.

SACHS.

Prognostic Significance of Tuberculous Cavities in the Lungs—M. FISHBERG, N. Y. Med. Jour., June 26, 1915.

In very acute forms of tuberculosis, cavitation is exceedingly rare. The prognosis is gloomy with or without localized destruction of pulmonary tissues. In adults such cases are rare, but in infants rapid cavity formation is seen at times, and the termination is almost

invariably fatal. In subacute forms of phthisis in which excavations are apt to form very rapidly, the prognosis is unfavorable, unless the cavity is rather small. In chronic phthisis, excavations even when extensive are compatible with a long and efficient life. On the whole cavities are an indication of chronicity of the tuberculous process in the lungs.

SACHS.

Tuberculous Meningitis and Tuberculosis of other Organs—W. STEINMEIER, *Virchow's Archiv*, Vol. CCXVI, Nos. 1 to 3.

A statistical study. Children are particularly prone to tuberculous meningitis. In nearly 45 per cent. of the cases the tuberculous meningitis was a part phenomenon of a generalized tuberculosis. In more than 7 per cent. of the cases there was a combination with urogenital tuberculosis.

MILL.

Syphilis and Tuberculosis—A. S. MACNALT, *Practitioner* (London), June, 1915.

Syphilitic affections of the lungs or glands may simulate tuberculosis of the lungs or glands. Syphilis of the lungs is stated by many authorities to be a disease of great rarity, while others advance the opinion that it occurs more frequently than has been supposed, but that it has been confounded with tuberculosis. The possibility of error is augmented by the fact that syphilis and tuberculosis may occur at the same time in the same subject.

SACHS.

Wassermann Reaction in Relation to Diagnosis—R. B. H. GRADWOHL, *Southern Med. Jour.*, June, 1915.

In the recognition of a latent case of syphilis, an obscure case, a tertiary case, the case that has had syphilis a long time ago, etc., the Wassermann reaction will often make the diagnosis where all else fails. Cases are legion that have gone the rounds and have been treated for everything but syphilis. Some there are that deny ever having had syphilis, denying either a primary sore or a secondary skin manifestation. Some lie and others don't know. Some are late congenital cases that naturally know nothing of their parents' infection. Possibly the most monumental sin that besets the profession to-day is its readiness to accept any statement from any patient regarding the history of a past syphilitic infection in so far as it has any bearing on the inclusion or exclusion of syphilis in explaining a present obscure disease. We are too prone to ask a patient whether he ever had syphilis, and on being told "no" to accept this as final proof that he cannot possibly now have the disease. We are also too much inclined to regard the patient's respectability as offering a bar to the existence of syphilis. We are too prone to think of syphilis as the last explanation instead of the first in seeking the cause of something obscure. Some of us are overawed by the apparent or real chastity of our patients, particularly of the female sex. Sex, age, social position, marital status, nothing should

stand in our way in investigation of disease. And there should be no hesitation once the diagnosis is made in telling the patient the real nature of his disease. It is in this class of patients that the Wassermann looms up as the great hope of diagnosis. The blood serum examination usually suffices to pin down the existence of a stomach or liver or heart or lung syphilis.

Luetin Test in Parasyphilis—D. M. ROSS, *Jour. Mental Sci.*, April, 1915.

The luetin reaction is a valuable addition to our diagnostic tests for syphilis. It is easily carried out by the clinician, is absolutely specific for the disease, and is occasionally positive in cases in which the Wassermann reaction is negative. Much states that when it is a question of ascertaining if the patient has ever been infected with syphilis, the luetin test is the more instructive, but when it is desired to know if the disease is still active, the Wassermann is the more helpful.

SACHS.

Wassermann Reaction in Malaria, Kala-Azar and Leprosy—W. D. SUTHERLAND and G. C. MITRA, *Indian Jour. Med. Research*, April, 1915.

Chronic malaria does not affect the Wassermann at all. One must wait a week until the patient's peripheral blood has been clear of parasites before having the blood tested. In 38 cases of kala-azar the Wassermann reaction was positive in 10, but only 2 of these cases gave a more than slightly positive reaction. In 34 undoubted cases of leprosy, 14 were of the anesthetic form; of these 4 gave a positive Wassermann reaction. Of the remaining 20 cases, 7 gave a positive reaction.

SACHS.

Döhle's Leukocyte Inclusions—H. REHDER, *Deutsches Archiv f. klin. Medizin*, Vol. CXVII, Nos. 1 and 2.

Typical leukocyte inclusions (trypanochetes), as originally described by Döhle, are only found in scarlet fever and, with very few exceptions, in no other diseases. Atypical inclusions, on the other hand, occur in almost all febrile affections. The true nature of these bodies is not as yet definitely known. It is not known if these inclusion substances are spirochetes or protozoa, either of which may be a possible causative agent of scarlet. The atypical leukocyte inclusions are probably only protoplasm particles.

WESTERN.

Positive Gruber-Widal Reaction in Dysentery—R. MAREK, *Wiener klin. Wochenschr.*, May 20, 1915.

Author noted a positive Gruber-Widal reaction in a number of cases which seemed to be dysenteric colitis in a clinical sense. In many cases it is not possible to obtain a bacteriological and serological diagnosis of dysentery. It may be surmised that some of the causative factors of dysentery are closely related, morphologically and biologically, to the typhoid bacilli. It is noteworthy that in some cases after preventive vaccination for typhoid the dysentery ag-

glutination, which became negative, is again rendered active. At any rate, a positive Gruber-Widal reaction is no longer a positive proof for the existence of typhoid fever if the clinical course of the disease does not point to this infection. MILL.

Dysenteric Rheumatoids—G. SINGER, Wiener med. Wochenschr., Feb. 8, 1915.

Among 600 cases of dysentery, author has met polyarthritic rheumatoids in 7 instances. These rheumatoids must be considered to be of metastatic origin; on account of their septic character they are related with the acute rheumatic polyarthritis. MILL.

Symptomatology and Diagnosis of Typhoid Fever—F. SCHULTZE, Deutsche med. Wochenschr., June 17, 1915.

Typhoid fever may be ushered in with chills. These, however, are of rare occurrence. The violent pains in the head and neck must be due in certain cases to the presence of encephalomyelomeningitis. Herpes may occasionally be noted in the beginning of typhoid fever. Author has had typhoid fever 42½ years ago, but still shows a positive Gruber-Widal reaction. MILL.

Involvement of the Kidneys in Relapsing Fever—L. JARNO, Wiener klin. Wochenschr., April 22, 1915.

Examinations of 170 cases. It was found that, as a rule, small amounts of albumin were already present on the first day of the first attack. On the second day the albumin content was increased to 0.5 to 1.5 per mille. The albumin continued in the urine until defervescence ensued. The sediment contained large numbers of granular casts, which disappeared together with the albumin. The second attack runs mostly a similar course. In later attacks the albuminuria and cylindruria are less marked. A permanent renal injury was noted in but one of the cases. In two instances hemorrhagic nephritis had been present. MILL.

Multiple Skin Infarcts after Measles—K. MORGENSTERN and G. B. GRUBER, Zeitschr. f. Kinderheilkunde, Vol. XII, Nos. 2 and 3.

After a case of measles there appeared skin infarcts and necroses on one side of the body. They were not due to embolism, but to localized thromboses of a number of arterial regions. MILL.

Chronic Progressive Polyarthritis—S. W. BOORSTEIN, Med. Rec., June 19, 1915.

This paper is a report of 105 cases of chronic progressive polyarthritis. Author concludes that the disease has no relation at all to rheumatism. The predominating etiological factor is distinctly an infection, either in the joint itself or at a distant point. The disease is not dangerous to life and is usually self-limited. SACHS.

Cholera—E. GILDEMEISTER and K. BAERTHLEIN, *Münchener med. Wochenschr.*, May 25, 1915.

Some of the conclusions reached by authors are as follows: Cholera vibrions die within a short time in a large portion of the cholera discharges. The vibrions, however, may remain viable in a not inconsiderable number of stools for a number of weeks, occasionally longer than 30 days. In the intestinal discharges of healthy germ-carriers cholera vibrions may also retain their viability for some weeks.

MILL.

RESPIRATORY AND CIRCULATORY ORGANS

Broncho-Pneumonic Pseudo Croup—E. SUÑER, *Jahrbuch f. Kinderheilkunde*, Vol. LXXX, No. 6.

The diagnosis of broncho-pneumonic pseudo croup is based upon the following characteristics: (1) Appearance of the initial catarrh (tracheitis and bronchitis); (2) absence of pseudo membranes in the nose, trachea, tonsils, larynx, etc.; (3) clinical symptoms of broncho-pneumonia together with laryngeal phenomena; (4) bacteriological examination. In dubious cases diphtheria antitoxin is recommended.

MILL.

Latent Syphilitic Infection of the Lungs—H. R. M. LANDIS and P. A. LEWIS, *Am. Jour. Med. Sci.*, August, 1915.

The diagnosis of latent syphilitic infection of the lungs must be made by exclusion. Thus if the symptoms and physical signs are those characteristic of tuberculosis and the sputum does not contain tubercle bacilli, or the progress of the case differs from that usually encountered in tuberculosis, the possibility of some other exciting cause should be thought of. Not only should the sputum be examined for organisms other than the tubercle bacillus, but in addition a Wassermann test should be made in every doubtful case. SACHS.

Hypertrophy of the Right Heart—J. BRET, *Progrès méd.*, June 27, 1914.

In all cases of hypertrophy of the right ventricle, it is not the lesions of the pulmonary parenchyma so much as the lesions of the vessels that play the pathogenic rôle. They are, in fact, the atheromatous lesions of the pulmonary artery, as described by Giroux, which is accompanied by more marked hypertrophy of the right ventricle, and sometimes less marked atheroma of the pulmonary artery is associated with mitral contraction. The clinical symptoms are: marked cyanosis, dyspnea, drowsy torpor, hepatic hypertrophy, albuminuria, acceleration or slowing of the cardiac rhythm, without arrhythmia, systolic pressure of 110 or 120 mm. Hg. (Riva-Rocci), diastolic pressure 90 to 95, polyglobulia and increased viscosity. These symptoms comprise the primary asphyxial syndrome.

ZIMMER.

Irregular Action of the Heart—C. WILSON, *Brit. Med. Jour.*, June 5, 1915.

Definite heart-block is always serious and the patient should be kept well within his limits, as his life is insecure. The young patient exhibiting sinus arrhythmia should not be debarred from any occupation or recreation suited to the general physique, nor should any treatment be prescribed except on other grounds. When there is no direct evidence of cardiac mischief, the irregularity due to extra systoles may be absolutely ignored. Where heart disease exists, patients showing this form of arrhythmia may be reassured and encouraged to exercise their activities to the extent of comfort. They need not live in fear of sudden death. Auricular fibrillation is very common in damaged hearts. A majority of the cases of heart failure exhibiting dropsy are associated with this condition. It can often be diagnosed from simply feeling the pulse. An absolute irregularity which persists is in itself sufficient. In heart-block sinus arrhythmia and extra systole, exertion or any influence which quickens the pulse tends to reduce or perhaps abolish the irregularity, while in fibrillation precisely the opposite effect is produced. Pulsus alternans often occurs only as a terminal phenomenon, and as such it has, when recognized, a grave prognostic significance. Paroxysmal tachycardia is sometimes fatal, but more often passes and leaves the patient but little worse until the next attack occurs. Auricular flutter is generally associated with sclerotic changes of advancing years. Ability to differentiate the rhythms will restore far more patients to activity than it will condemn.

SACHS.

Transitory Complete Cardiac Irregularities—K. FAHRENKAMP, *Deutsches Archiv f. klin. Medizin*, Vol. CXVII, Nos. 1 and 2.

There are marked cardiac irregularities which are transitory, lasting generally but a few hours, in which the nerve influence is often clearly noticeable. This nerve influence consists in an increased vagus irritability. These arrhythmias appear not always in the frame of the same clinical picture. The symptom-complex is apt to change in the same patient. It is clinically of importance that extra systolic arrhythmia be differentiated from perpetual arrhythmia.

WESTERN.

Alternation of the Pulse—P. D. WHITE, *Am. Jour. Med. Sci.*, July, 1915.

True alternation of the radial pulse has been found in 71 of 300 cardiac and cardiorenal patients examined with the sphygmograph by the writer at the Massachusetts General Hospital. It has occurred as commonly as auricular fibrillation. The relationship of the degree of alternation to prognosis was as one would anticipate: the greater the degree, the shorter the life after the pulse discovery.

SACHS.

Permanent Bradycardia—COTTIN, Archives Maladies du Cœur, des Vaisseaux et du Sang, June, 1915.

Chronic slowness of pulse, due to auriculo-ventricular dissociation, is not always the result of a lesion of the bundle of His, as has long since been demonstrated. The hitherto accepted notions that paroxysmal occurrence of attacks of bradycardia were invariably of nervous origin, and that auriculo-ventricular dissociation, combined with a lesion of the bundle of His, was the cause of permanent bradycardia, have been modified by the recent researches of Rathéry and Lian, who have demonstrated in the Paris hospitals two cases of permanent bradycardia of nervous origin. Still more recently Souques and Routier have published observations of three cases of Adams-Stokes disease, also of nervous origin. ZIMMER.

Patency of the Ductus Arteriosus—T. W. GRIFFITH, Quart. Jour. Med. (London), April, 1915.

Two examples of the patency of the ductus arteriosus occurred in patients in whom the affection was recognized during life. One of these succumbed to an infective endocarditis, and the diagnosis was verified on the post-mortem table. In the other the condition was probably associated with some further anomaly of the great vessels, and with a developmental error which gave rise to cyanosis, for this is a symptom which is not often met with in uncomplicated cases of patency of the arterial duct. In the first case there was heard at the inner end of the second left intercostal space a loud systolic bruit, followed by a very loud diastolic bruit, which was audible a considerable distance outwards along the second space and was not transmitted along the left edge of the sternum. These two sounds produced as it were one continuous bruit with systolic and diastolic increments. SACHS.

The Rôle of Syphilis in Hypertensive Cardiovascular Disease—H. F. STOLL, Am. Jour. Med. Sci., August, 1915.

The results of author's investigation is summarized thus: Syphilis is the underlying or basic factor in a much higher percentage of hypertensive cases than has hitherto been realized. Of 50 individuals studied 90 per cent. either gave a positive Wassermann or luetin test, or were known to have had lues, or had children with hereditary syphilis. Nineteen were from cardiovascular families. In 17 of them either one or both tests were positive. One of the two with negative reactions is surely specific. This strongly suggests the existence of what might be termed "familial cardiovascular syphilis." It would seem that hypertensive disease is one of the most common—possibly the most frequent—of the so-called "late" manifestations of hereditary syphilis. Apoplexy and sudden cardiac death occurring in middle life are almost always due to syphilis, and it cannot be considered a negligible factor even in the aged. The re-

maintaining parent and the children of individuals dying a cardiovascular-renal death in middle life should be tested for syphilis, as they are infected in a high percentage of the cases. SACHS.

Cardiac and Vascular Diseases in the War—E. ROMBERG, *Münchener med. Wochenschr.*, May 18, 1915.

Persons with sclerotic, not normally functioning arteries are, comparatively, more frequently affected with cardiac insufficiency than individuals with normal blood vessels. However, exhausted and reconvalescent individuals or those with latent fever who are troubled with cardiac disorders, even those exhibiting accidental sounds, must not necessarily be affected with heart disease. Examination of heart and pulse often do not suffice to form a definite opinion about the condition of the heart. In order to come to a definite understanding, it is always essential that the condition and behavior of all the other organs that stand in connection with the circulation, and the previous medical history of the individual be taken into due consideration. MILL.

Media Calcification and Atherosclerosis—J. G. MÖNCKEBERG, *Virchow's Archiv*, Vol. CCXVI, Nos. 1 to 3.

Calcification of the media and atherosclerosis should be differentiated between. Neither the degree nor the extent of a peripheral calcification of the media justifies, without additional reasons, the assumption of an atherosclerosis of the central vessels. Both processes may, however, be found associated. MILL.

A Remarkable Reflex Phenomenon in an Aneurism of the Femoral Artery—WIGDOROWITSCH, *Deutsche med. Wochenschr.*, June 17, 1915.

Pressure upon the femoral artery beneath Poupart's ligament in an injured leg caused a decline of pulse-frequency from 72 to from 42 to 45 pulsations per minute. The pulse-frequency was not diminished in the healthy limb. MILL.

ALIMENTARY TRACT

Gastric Analysis—T. HEATON, *Brit. Med. Jour.*, April 24, 1915.

The interpretation of the figures obtained by gastric analysis after a test meal is by reason of the number of factors involved a matter of great complexity. A means of standardizing results is afforded by adding to the meal a fixed proportion of some inert substance, such as urea, whose proportion can be estimated subsequently in the gastric content. It is then possible to arrive by calculation at an approximation of the actual composition of the secreted gastric juice, as opposed to those of the mixture of this with the remnants of the fluid of the meal. By this method it is possible also to eliminate the disturbing influences of an abnormally rapid or an abnormally slow rate of emptying of the stomach into the duodenum. The condi-

tions of hyperacidity and hypoacidity can be differentiated from those respectively of hypersecretion and hyposecretion. SACHS.

Clinical Use of Water Meal—A. E. AUSTIN, Bost. Med. and Surg. Jour., June 10, 1915.

The patient is given, on the evening before he is examined, a meal of meat, potato, bread, butter, rice and raisins, and the next morning, on a fasting stomach, 350 c.c. of water is administered. Twenty minutes later, the extreme limit at which the water normally leaves the stomach, it is removed. The water meal has the advantage of readily allowing one to see whether or not there is any residue left from the Riegel meal. Lactic acid, blood and bile are also much more readily detected in the absence of a mass of partly digested food. The water meal is especially convenient for dispensary patients. SACHS.

Bismuth Pills in Fluoroscopic Examination of the Infant's Stomach—A. F. HESS, Am. Jour. Dis. Child., June, 1915.

Author used keratin coated bismuth pills having definite circumferences. His purpose was to furnish a simple means of gauging the size of the pylorus and of judging whether this sphincter was normally patent or not. By fluoroscopy it was found that under normal conditions objects do not leave the stomach in direct ratio to their size; that, in fact, larger objects are apt to be propelled into the intestine more quickly than smaller ones. Thus it is possible that food which has been insufficiently masticated may remain in the stomach for a shorter period rather than for a longer period than food that has been thoroughly comminuted. Probably this is frequently the case. The pills were delayed in their passage through the pylorus when the infant was placed on the left side, and were hastened in their passage by placing the infant on the right side. In cases of pylorospasm there was a retardation in the passage of the pills from the stomach into the intestine. The degree of this delay varied in accordance with the degree of obstruction. SACHS.

Fatigue Dyspepsia—G. RANKIN, Brit. Med. Jour., June 19, 1915.

Fatigue dyspepsia is characterized in every case by an irritability or exhausted state of the nervous system, the result of over strain. It is met with almost exclusively in those whose labors are intellectual or originative. Every case is accompanied by the landmarks of the neurasthenic state. The appetite is not necessarily seriously impaired, but the patient becomes suspicious of one thing after another in his daily dietary, and cuts it out of his menu in the hope that he will thereby find relief to the miserable discomfort which he comes to realize will inevitably succeed each meal. This discomfort comes on 2 to 4 hours after the taking of food, and declares itself by a gnawing and burning sensation in the stomach, followed by a feeling of oppressive distension, with a commanding desire to

find relief in constantly repeated eructations or even active vomiting. The taking of more food gives him immediate though temporary immunity from his trouble. He adopts the plan of having certain food at his bedside in order to secure such comfort. SACHS.

Syphilis and Gastric Symptoms—T. BRUGSCH and E. SCHNEIDER, *Berliner klin. Wochenschr.*, June 7, 1915.

Besides the formation of gummata, the following phenomena are characteristic of syphilis: (1) The fact that gastric ulcer is mostly associated with diminished HCl secretion in tertiary syphilis; (2) the frequent achylia in lues, which in all probability is due to a chronic gastritis; (3) sensory irritation phenomena, (2) root-neuritic zones of the middle dorsal segments, which certainly do not stand in relation to the stomach, but are thought to be stomach diseases by the patients; (b) sensory irritation phenomena, probably due to the vagus; (4) motor irritation phenomena outside of typical zones; (5) states of supersecretion, on the other hand, seem not to be characteristic of syphilis. The combination of violent pain and supersecretion, however, is not rare, and points to gastric ulcer without any connection with syphilis. MILL.

Physical Signs Referable to the Diaphragm—R. DEXTER, *Am. Jour. Med. Sci.*, August, 1915.

Inflammation or irritation of the pleural or peritoneal surface of the diaphragm does not give rise to local symptoms. The pain resulting from such processes is referred upward along the phrenic nerves to the third or fourth cervical segments or downward along the sixth or twelfth intercostals into the lower dorsal segments. The pain is usually accompanied by tenderness and hyperesthesia or hyperalgesia of the skin. The recognition and interpretation of these signs may be of considerable importance in differential diagnosis between intrathoracic or intraperitoneal disease, in the absence of any signs in the lungs. When a part or the whole of the diaphragm is forced downward the contraction of the diaphragm exerts a more powerful inward pull along the line of its attachments. This is especially marked when the anterior portion of the diaphragm is depressed. This will result in a lessening of the outward excursion of the subcostal angle, or an actual retraction along the line of diaphragmatic attachment. Conversely any condition which lifts the diaphragm upward lessens the strength of the inward pull of the contracting diaphragm, with the result that the normal outward movement of the costal margins will be increased. The presence of a retraction or of an abnormal outward flaring of the subcostal angle will often be of aid in the explanation of obscure diseases of the viscera which lie immediately above or immediately below the diaphragm, especially in pericardial effusion or in subphrenic abscess.

SACHS.

Gastrocoloptosis in Radiologic Respects—O. STRAUSS, *Deutsche med. Wochenschr.*, June 17, 1915.

The article represents a review of the book of Rovsing on this question. This book offers nothing new concerning the clinical picture of gastrocoloptosis. A surgical interference is not advocated unless continued observations show the superiority of this mode of treatment. In uncomplicated instances of gastropotosis the X-ray examination determines the diagnosis. MILL.

Lymphocytosis, a Sign of Constitutional Disturbance in Chronic Affections of the Gastrointestinal Tract—J. KAUFMANN, *Mitteilungen a. d. Grenzgebieten d. Medizin u. Chirurgie*, Vol. XXVIII, No. 3.

In 60 per cent. of chronic diseases of the alimentary tract (140 cases) lymphocytosis was noted. This lymphocytosis may be an expression of an abnormal constitution (status hypoplasticus). Author believes that a constitutional anomaly may form the basis on which functional alimentary disturbances may develop. This, he continues, occurs mostly through a disorder of an internal secretion to which patients of this class are particularly predisposed. MILL.

Improved Phenolphthalein Reaction for the Demonstration of Occult Blood in the Feces—J. BOAS, *Deutsche med. Wochenschr.*, May 13, 1915.

Author describes his "Phenolphthalein ring test." The feces are extracted by glacial acetic acid-alcohol (5 drops glacial acetic acid to 15 to 20 grams alcohol). The phenolphthalein reagent (15 drops) is placed into a test tube; to this is added 5 to 6 drops of hydrogen peroxid (3 per cent.) and 2 c.c. absolute alcohol. The mixture is then well shaken. The fecal extract is then filtered through a funnel into the test tube in such a manner that the filtrate flows slowly down the side of the tube into the reagent. In the presence of blood coloring matter a ring, light or dark red, is formed. MILL.

Referred Pains—G. DURAND, *Progrès médical*, July 25, 1915.

From an exhaustive study of the semeiologic value of referred pains, author has formed the conclusion that, while referred pains are not, as some authors have asserted, "almost pathognomonic of a lesion of the pylorus," and do not, of themselves, indicate a definite and localized disease of the pylorus and duodenum, arising from whatever cause, they are of incontestable value in diagnosis, but merely as a signal of alarm. Examination of the patient alone should ascertain the cause of the pyloric affection and therapeutic treatment may aid in establishing a diagnosis in difficult cases. Author does not admit that the syndrome of "referred pains" absolutely indicates pyloric contraction from a lesion of the gastric mucous membrane, but considers that functional pyloric disturbances are due to a multiplicity of causes. Innervation of the sphincter may result from various disturbances, whether of peripheral, central, or reflex origin, and all these causes should be taken into con-

sideration as etiologic factors of the pyloric cramp, and of the syndrome of referred pains, which are its expression. As a fact, referred pains of gastroduodenal origin are present in the great majority of cases, the attack occurring at regular hours, and it is this, rather than their topography and irradiation, that gives them a nosologic individuality. In practice, pain in the gall-bladder and pancreas, which is much rarer, may be confounded with "referred pains" in diagnosis. But although cholelithiasis and pancreatic lithiasis, or pancreatitis, may frequently give rise to the syndrome of inveterate "referred pains," confusion in diagnosis need not occur if the primordial or accessory symptoms of the patient be carefully studied. ZIMMER.

New Theory of the Causation of Enterostasis—A. KEITH, West London Med. Jour., July, 1915.

In passing along the alimentary tract food is propelled through a series of zones or segments, each furnished with its own pacemaker and its own rhythmical contractions. Irregularities may occur in the nodal and conducting system of the alimentary tract—irregularities of the same kind which are known to occur in the heart. When such irregularities or blocks do occur, we find them at the points where one rhythmical zone or area passes into the succeeding zone, as at the junction of the esophagus and stomach, at the gastroduodenal junction, where the duodenal zone passes into the jejunal and where the jejuno-iliac passes into the ileo-colic. In order to have an orderly propulsion of food along the whole length of the alimentary canal, those various rhythmical zones must be closely coordinated in their action. Bayliss and Starling observed that distension of the duodenum inhibited the action of the ileum. Thus one can understand how stasis in the great bowel may be followed by ileal stasis, duodenal or gastric stasis, or how a disturbance of the conductivity or excitability of any of the rhythmical zones may ultimately give rise to stasis in all. SACHS.

Intestinal Occlusion—A. MATHIEU, Archives Maladies de l'Appar. Digest. et de la Nutrition, June, 1914.

In incomplete stenosis of the pylorus, during paroxysms of pain, a considerable amount of fluid accumulates in the stomach; this is due to glandular hypersecretion and peristaltic contractions, visible to the naked eye, and still more apparent under radioscopic examination, showing the strenuous efforts made by the muscular pouch to overcome the obstacle. It appears that at the time of the painful pyloric cramp there occurs considerable hypersecretion and exaggerated peristalsis, and sometimes even anti-peristaltic contractions. Frequently there is abundant vomiting of a liquid which, should the pyloric lesion be of an ulcerating character, contains an increased amount of hydrochloric acid, which is the chemical characteristic of

gastric hypersecretion. In incomplete stenosis toward the hepatic angle of the colon, there frequently appears a syndrome analogous to that named after Bouveret, of Lyons, in which there are also paroxysms of pain, with dilatation of the cecum, which may be perceived outwardly. The dilated cecal pouch also frequently betrays peristaltic contraction occurring synchronously with the paroxysm of pain, and examination shows the presence of a greater or less amount of fluid in the dilated cecum. This syndrome is to the cecum what the pyloric attack with hypersecretion is to the stomach. It has a physio-pathologic analogy to Koenig's syndrome, which deals, however, only with the small intestine. Should the stenosis affect the initial portion of the jejunum, it may be difficult to distinguish between intestinal dilatation and dilatation of the stomach from pyloric stenosis. As washing out the stomach leaves below a second, dilated and pulsating pouch, it was extremely difficult to make a diagnosis between stenosis of the jejunum and gastric biloculation, before radioscopy came into use. Identical reaction occurs when the stenosis is much lower down, near the end of the ileum. There is present also painful colic, dilatation and distension of the intestinal flexures, the peristaltic contractions of which are apparent at the moment of the colic, and there may be a considerable accumulation of hypersecretion fluid and of stasis in these dilated flexures, which may be returned or driven back into the stomach by the anti-peristaltic contractions. The abundant vomiting which occurs in stoppage of the small intestines also occurs in stenosis of the colon when the ileocecal valve is forced. Author considers that abdominal clapotage and false ascites is of great symptomatic value in the diagnosis. It is often impossible for some time to decide whether it is a case of intestinal stoppage or marked gastric dilatation from pyloric stenosis.

ZIMMER.

Diagnosis of Colon Cancer—J. BURKE, N. Y. State Jour. Med., July, 1915.

In cases of unexplained loss of weight and diminished muscular strength, with secondary anemia in any adult above forty years, particularly if gastrointestinal symptoms are present, cancer of the colon should be carefully considered. Where a tumor is present in any of the four corners of the abdomen colon cancer must be thought of. When peritoneal friction sounds are heard over the tumor it speaks positively for its intraperitoneal origin. In sudden profuse hemorrhage from the bowel the colon should be diligently investigated for cancer, particularly the sigmoid flexure. When an adult complains of colicky pains in the abdomen, particularly when accompanied by disturbances of bowel function, colon cancer should be thought of as the probable cause. In cases of suspected acute appendicitis in elderly people, cancer of the cecum must not be lost sight of in our diagnostic deliberations. In all cases where there is

the slightest suspicion of colonic derangement the X-ray should never be omitted in the examination. In all cases of suspected cancer of the bowel, X-ray examination should always be made. The X-ray is the greatest aid modern science gives us in the differential diagnosis of colon cancer.

WESTERN.

Pancreatic Infantilism—B. BRAMWELL, *Edinburgh Med. Jour.*, May, 1915.

Pancreatic infantilism is characterized by arrested bodily and sexual development; there is no mental defect or deformity or structural defect of the bones; there is no visceral derangement or disease except chronic diarrhea, flatulent distension of the abdomen and defective or arrested pancreatic secretion. The latter is probably due to a chronic pancreatitis. In some cases the condition of diarrhea and infantilism is cured by the administration of pancreatic extract.

SACHS.

Biliary Lithiasis—M. A. GILBERT, *Jour. Méd. Franç.*, April 15, 1914.

Gall-stones may make their way through the biliary ducts and reach the intestines, or, on the other hand, finding it impossible to pass through the ducts, may fall back into the gall-bladder. The first proceeding induces the ordinary hepatic colic, with expulsion of the gall-stones; the second causes gall-bladder colic, without expulsion of the gall-stones. Both forms of colic occur about 3 to 4 hours after a meal, and present an analogous clinical picture. In some cases they may be distinguished from one another by a biliary tumor or swelling, indicating the occlusion of the gall-bladder; this may disappear rapidly when the attack subsides, or may, in exceptional cases, last a week. Certain negative symptoms should never be overlooked, such as the absence of icterus, hepatalgia and hepatomegaly, and the absence of biliary concretions in the stools should be specially noted. The attack is usually sudden, sometimes continuous, sometimes paroxysmal, and may last from a few hours to several days, or even weeks. It is generally less painful, but lasts longer than ordinary hepatic colic with expulsion. In a lithiatic patient gall-bladder colic may alternate with ordinary colic, but usually one form prevails. The intervals vary, but are usually shorter than in the case of expulsive colic. A course of cholagogic treatment may induce veritable liver complaint. In some instances, gall-bladder colic may recur all through the life of a patient. The attacks generally cease at a given time because the stones become immovable in the gall-bladder; sometimes one or two attacks of colic, with expulsion of the gall-stones, will rid the bladder of them, but there are apt to be complications. A stone may be imprisoned in the bladder, forming a permanent gall cyst, which often turns into cholecystitis. Then, after biliary and peribiliary phlegmons, the gall-stone may penetrate into the most abnormal passages. The diagnosis is often very difficult if the malady is recent, and there is no

cholecystitis. One thinks of poisoning, of gastric pain, of a gastric attack due to tabes. If there is a cholecystitis, the diagnosis is localized in the biliary passages, and only as the attack develops, and as one discovers gall-stones in the stools, can one determine whether or not it is an expulsive attack. Recent cases are, of course, less readily diagnosed.

ZIMMER.

NERVOUS SYSTEM

Vagotonia—W. LUBLINSKI, *Berliner klin. Wochenschr.*, May 7, 1915.

The clinical picture of vagotonia consists of contracted pupil, salivation, sweating, acrocyanosis, pronounced dermographia, bradycardia and respiratory arrhythmia, cramps in the upper tracts and the abdominal organs, probably also in the vesical sphincters. Atropin influences the vagotonia by reducing the irritability of the vagus terminals.

MILL.

Vegetative Nervous System and Abdominal Diseases—A. THIES, *Mitteilungen a. d. Grenzgebieten d. Medizin u. Chirurgie*, Vol. XXVIII, No. 3.

On the hand of a large number of clinical cases author again confirms the fact that abdominal affections may give rise to symptoms of the vegetative nervous system. The examination which includes phenomena on the part of the eyelids and pupils (contraction, dilatation) shows a certain regularity of the eye phenomena in accordance with the localization of the abdominal disease. After disappearance of the abdominal symptoms those of the eyes are diminished or disappear altogether.

MILL.

Multiple Neuritis complicating Typhoid Fever—T. A. CLAYTOR, *Am. Jour. Med. Sci.*, May, 1915.

Author collected 25 cases of multiple neuritis complicating or following typhoid fever, which with his own case make a series of 26. Symptoms of the condition were recognized in 14 instances during the course of the fever, while 12 cases were recognized after the fever had subsided. Pain was present 17 times, absent 9 times. Paralysis was noted 25 times; in 1 case it was not mentioned. The duration of the condition varied from 3 to 14 months. Unqualified recovery took place in 11 cases, improvement in 7, death in 2, and the result was not given in 6 cases.

SACHS.

Visual Fields in Brain Tumor—H. CUSHING and C. B. WALKER, *Brain*, Vol. XXXVII.

In a series of 454 cases classified as tumor of the brain, there have been 101 in which the lesion was of hypophysial or parhypophysial origin, and in 81 of these cases, chiasmal involvement lead to deformation of the fields of vision. These deformations tended at the time of admission to be bitemporal in 26 cases, homonymous in 12, were unclassified in 8 cases, and in the remaining 35 showed blind-

ness in one or both eyes, making it difficult to tell in which group—bitemporal or homonymous—they belonged. Detailed perimetry with small test objects of serial sizes, particular attention being paid to the shading off of the upper temporal peripheries and to the presence of relative paracentral scotomata in the same quadrant, is advocated for patients with pituitary disease in order that stages of hemianopsia antecedent to those usually recognized may be detected.

SACHS.

Abderhalden's Protective Ferments in Psychiatry—F. SIOLI, *Archiv f. Psychiatrie u. Nervenkrankheiten*, Vol. LV, No. 1.

The results obtained by Abderhalden's method in psychiatry are not sufficiently univocal that they can be employed in the diagnosis, prognosis or pathogenetic basis. Again, the method is subject to many sources of error. Before the method finds clinical application it has to be thoroughly tested.

WESTERN.

Hemolysin Reaction of Weil-Kafka in Psychiatric Diagnosis—E. L. BRÜCKNER, *Archiv f. Psychiatrie u. Nervenkrankheiten*, Vol. LV, No. 1.

As a rule, the reaction is positive in progressive paralysis. In paralysis, suspected for clinical reasons, the reaction is a valuable adjuvant of the clinical diagnosis.

WESTERN.

Physical Manifestations of Dementia Precox—B. HOLMES, *Lancet-Clinic*, July 17, 1915.

The morphologic microscopic picture of the testicle in dementia precox is conclusive of a progressive destructive process or affection involving especially the seminiferous tubules, but also modifying the appearance of the chromaffin cells or the cells of Leydig. The brain in dementia precox is hydrocephalic, and the brain weight is too great both for the size of the skull and the size and weight of the body. Catatonia is a condition of wet brain, and if we interpret Nissl's last thesis correctly it would not be irrational to treat it by compression.

WESTERN.

Spirochaeta Pallida in Dementia Paralytica—J. MCINTOSH and P. FILDES, *Brain*, Vol. XXXVII.

The brains of 7 cases of dementia paralytica were examined and in 6 of these spirochetes were found by the dark ground method, although when sections were cut, the organisms were only detected in 3. The spirochetes were always confined to the gray matter; in one case only was a single specimen seen in the meninges.

SACHS.

Paranoid Psychoses in Old Age—SEELERT, *Archiv f. Psychiatrie u. Nervenkrankheiten*, Vol. LV, No. 1.

Report of 12 pertaining cases on the basis of which a description of paranoid psychoses of old age is given. On account of their symptomatology and course these psychoses represent a special group. The one symptom common to all these psychoses is the pres-

ence of delusions. The patients believe themselves to be wronged, molested and persecuted; they are suspicious and distrustful. Misinterpretation of their observations and experiences, especially misconstructions of disease processes of body and mind, play an important part. The frequent paroxysmal pains and annoyances, as dizziness, ringing in the ears, fear, sense of heat, headache, sleeplessness, etc., probably ensue upon an organic substrate. These symptoms, the age of the patient and their frequent concurrence with cardiac and vascular disturbances, point to the existence of arteriosclerotic changes. All these phenomena are explained by the patients to be the consequence of molestations on the part of their entourage, and are thought by them to be due to poisoning with vapors, spoiled food, medicaments, etc. The anamnesis evinces that these patients were always more or less excitable and easily affected psychically, that they were always prone to misinterpret or misconstrue, and that these traits were always noticeable in the character and temperament of the patients. Later on in life these characteristics become more pronounced and then constitute the psychosis. It is, therefore, probable that the paranoid psychoses of old age are an individual reaction form of an endogenous, slowly progressive organic process.

WESTERN.

New Symptoms in Amaurotic Family Idiocy—I. H. CORIAT, Bost. Med. and Surg. Jour., July 1, 1915.

Author has noted in some cases of amaurotic family idiocy the following new symptoms, which are not as a rule included in the classical description of the disease: Hydrocephalus; bulbar symptoms—drooling of saliva, choking spells, difficulty in swallowing, and attacks of apnea; nystagmus, hypotonia, and abnormal reflex phenomena. Increased reflex action to sound and touch are among the most interesting phenomena of this disease.

SACHS.

URINARY ORGANS—MALE GENITALIA

Determination of Retention Nitrogen in the Blood, a Method for Testing Renal Function—H. HOHLWEG, *Mitteilungen a. d. Grenzgebieten d. Medizin u. Chirurgie*, Vol. XXVIII, No. 3.

In monolateral affection of the kidneys the retention nitrogen in the blood is never increased. This is also the case when the individual possesses but one kidney which is sound. When, however, the renal affection is bilateral, or when the single kidney present is affected, the retention nitrogen is always increased.

MILL.

Occult Hemorrhage from the Renal Pelvis—EICHHORST, *Zentralblatt f. innere Medizin*, No. 12, 1915.

The demonstration of cells of blood pigment (large round cells with blood pigment) in the urinary sediment is of great significance

in the diagnosis of calculi in the pelvis. These cells are derived from colorless blood corpuscles.

WESTERN.

Symptomless Renal Hematuria—D. NEWMAN, Brit. Jour. Surg., April, 1915.

The absence of symptoms and of physical signs beyond the presence of blood in the urine is characteristic of small tumors in the pelvis or in the medullary substance of the kidney. It may be present for a long period in such cases before pain or any other symptom is complained of. Aneurism, occupying the pelvis and rupturing into it, may cause hematuria and the bleeding may persist for months before any other symptoms develop. The hemorrhage may be fatal before other signs show themselves; but usually the bleeding is small in amount and gradually increases. In a few cases of renal calculus blood may appear in the urine unattended by subjective evidence of disease. Symptomless hematuria in tuberculosis is a more common occurrence than is generally suspected. It is met with in young persons or in children.

SACHS.

Ureteral Calculi—J. T. GERAGHTY and F. HINMAN, Surg. Gynecol. and Obstet., May, 1915.

Except in rare instances, the symptoms of ureteral calculi are not diagnostic and are insufficient to definitely determine either its presence or position. In the most expert hands a surprisingly large percentage of calculi may not be determined by röntgenography. By means of collargol ureterograms a calculus will occasionally be detected which was not revealed by the simple röntgenogram. The employment of the waxed-tipped catheter is by far the most accurate method for the detection of ureteral calculi, and this method should be in more general use. In 6 out of 36 cases, it has located a stone in which repeated röntgenograms were uniformly negative.

SACHS.

Internal Aspect of Prostatic Suppuration—H. BROOKS, Med. Rec., July 17, 1915.

General symptoms of a very striking and confusing character may develop in cases of prostatic suppuration. Prostration is the most impressive of these general manifestations of the disease. Mental depression, temperature elevation, profuse and drenching perspiration, slow pulse and a hypoleukocytosis may lead one to suspect the presence of typhoid fever. Rectal examination of the prostate will decide the diagnosis, but it must be remembered that prostatic suppuration may exist without any localizing symptoms.

SACHS.

Hematogenous Tuberculosis of the Prostate—M. SIMMONDS, Virchow's Archiv, Vol. CCXVI, Nos. 1 to 3.

In 11 per cent. of the cases of tuberculosis of the prostate the disease appeared to be hematogenous. The affection may appear in two forms, (1) as an excretion (Ausscheidungs) tuberculosis, (2) as

an interstitial, metastatic tuberculosis. The first form preponderates greatly.

MILL.

FEMALE ORGANS OF GENERATION—PREGNANCY— PARTURITION—INFANTS

Pathology of the Secretion of the Mammæ—P. LINDIG, *Zeitschr. f. Geburtshilfe u. Gynäkologie*, Vol. LXXVI, No. 3.

A non-physiological secretion of the mammæ is much more frequent than is generally assumed. Author found repeatedly colostrum and colostrum-like secretion in tuberculous nulliparis, in old patients with carcinoma, etc. It is possibly the destruction process as such which, by permitting an influx of protein decomposition products into the blood current, stimulates the activity of the mammæ. Reflex stimulation within the endocrinal glands may be a causative factor of the pathologic secretion of the mammæ.

MILL.

Benign Neoplasms of the Breast in Women—E. M. MOSHER, *Woman's Med. Jour.*, July, 1915.

In instances of benign neoplasms of the breast the patient may be a subject of hypothyroidism. In cases of this kind the thyroid activity should be investigated by administering thyroid extract. In other instances of benign growths the gastrointestinal canal should be carefully examined. The influence of enteroptosis and intestinal stasis upon the production of breast neoplasms should never be minimized.

WESTERN.

Prolapsus of the Uterus—H. J. BOLDT, *Am. Jour. Obstet. and Dis. of Women and Children*, 1915, No. 6.

Speaking on the relation of the other pelvic organs in instances of uterine prolapse, author says the following: When one does a vaginal hysterectomy or any other surgical intervention which necessitates separation of the bladder from the cervix, it must be obvious that, in cases of marked prolapsus the bladder too must be displaced, since the connective tissue between the bladder and the cervix—save in very exceptional instances—is quite firm, and therefore does not readily give way itself. Therefore it follows that the bladder descends with the cervix, and in this way forms a cystocele. Consequently it follows that, in marked instances of this kind, we may have residual urine after the act of voluntary micturition has been completed. But it is not only the bladder which descends. In marked instances the urethra also becomes dislocated at its bladder end. A similar dislocation takes place of the anterior rectal wall forming a rectocele, which has, however, as the underlying cause an injury of the soft parts during parturition. Whether the adnexæ become displaced in injuries accompanying uterine descent depends upon the position of the uterine body. If there be but a moderate

descent of the uterine body and principally an elongation of the cervix, the adnexæ are not likely to become displaced downward at all; but, necessarily, if the uterine body also descends markedly, the adnexæ (tubes and ovaries) must likewise follow the body to a greater or less extent. In instances of very marked uterine prolapse with descent of the entire vagina, the entire pelvic floor becomes so relaxed that it is evident that it can offer no support for the pelvic organs. SACHS.

Symptomatology of Corpus Luteum Cysts—J. HALBAN, *Zentralblatt f. Gynäkologie*, No. 24, 1915.

Report of 9 cases, of which 2 were complicated by gravidity. Occasionally cysts are developed from the corpus luteum. These cysts possess the property of the corpus luteum to inhibit menstruation. For this reason amenorrhea exists in the presence of corpus luteum cysts. This property is of import in the diagnosis. An exact diagnosis is essential in the pertaining cases, as cysts of this character may disappear spontaneously and do not need to be operated upon. Alternating cysts, i. e., alternating formation of cysts in both ovaries, disappearing spontaneously, must be considered to be corpus luteum cysts. These cysts are thin-walled and burst readily, even when the bimanual examination is made with the greatest of care. The cysts may also ensue during pregnancy. Their removal need not necessarily interrupt the pregnancy. MILL.

Ovarian Sarcomata in Children—T. T. HIGGINS, *Brit. Jour. Children's Dis.*, June, 1915.

Three cases are recorded which illustrate certain aspects of the disease, namely, the insidious onset, with unexplained colicky pains, later the appearance of a mobile lump, with possibly some precocious menstruation, the steady enlargement of the abdomen with pain and fever, the result of an adhesive peritonitis, occurring on the surface of the growth, and finally the tendency to spread, by direct extension along lines of adhesion rather than by metastases. SACHS.

Toxicity of Urine, Serum and Milk (Colostrum) during Pregnancy, Labor and Puerperium—P. WERNER and E. KOLISCH, *Archiv f. Gynäkologie*, Vol. CIII, No. 1.

The urine of healthy non-pregnant women is non-poisonous. Their serum is also non-toxic. The urine of healthy pregnant women is poisonous. In pregnancy-nephritis the toxicity of the urine is increased. In eclampsia the toxicity of the urine is reduced during labor, but increases immediately after it has taken place. The serum of healthy pregnant women is toxic. This is not the case with the serum of non-pregnant women. In pregnancy-nephritis the serum toxicity is also augmented. The serum of eclamptic women is, as a rule, poisonous before delivery has taken place, provided no edema is present. In the presence of edemas the serum is non-toxic. Colos-

trum exhibits a very small degree of toxicity. The toxicity of the milk is most marked on the third day of the puerperium. There is in this respect no difference between nephritic and eclamptic lying-in women. Edema liquid of nephritis is poisonous; it is non-poisonous of eclamptics (pregnant and puerperients), but poisonous of eclamptics in labor. MILL.

Cholelithiasis and Pregnancy—J. A. AMANN, *Monatsschr. f. Geburtshilfe u. Gynäkologie*, Vol. XXXII, No. 1.

During pregnancy and in the puerperium gall-stone colic occurs rather frequently. A case of the author exhibited such alarming symptoms that cholecystectomy had to be performed during pregnancy. The case was very complicated, as the appendix was adherent to the gall-bladder and as there was an inflamed fibrous tumor where the gall-bladder was grown to the abdominal wall. MILL.

Epilepsy and Pregnancy—C. MEYER, *Archiv f. Psychiatrie u. Nervenkrankheiten*, Vol. LV, No. 2.

It is only the more recent literature upon the subject of epilepsy and pregnancy that is of real value. The older literature did not differentiate between eclampsia and epilepsy. Pregnancy may provoke epileptic phenomena. It frequently influences the various manifestations of epilepsy in a favorable as well as in an unfavorable manner. WESTERN.

Circulatory Disturbances in the Newborn—A. v. REUSS, *Gynäkologische Rundschau*, Vol. IX, No. 1.

The icterus of the newborn depends in the main upon a hyperbilirubinemia, which is caused by an insufficiency of the youthful liver cell. The albuminuria of the newborn seems to be due to circulatory disturbances, not unlike those which give cause to orthotic or lordotic albuminuria. In a similar manner the congestive hyperemia, which is rather physiologic in the newborn, may be explained. Congestive hemorrhagia is but a continuation of the process that stands at the bottom of the congestive hyperemia. MILL.

The Production of Icterus Neonatorum—T. HEYNEMANN, *Zeitschr. f. Geburtshilfe u. Gynäkologie*, Vol. LXXVI, No. 3.

The causation of icterus in the newborn is in the first instance dependent upon a perverse and incomplete function of the liver cells. These cells are not able to perform the increased amount of necessary work with which they are confronted in the first days of life. The production of the icterus is enhanced by the congestive state of the liver and the pronounced disintegration of the red blood cells ensuing at this period. The cause of this disintegration is not definitely known. It is probably due to an increased activity of the stellate cells of Kupffer, which would indicate that it is the liver that gives rise to the destruction of the red cells. MILL.

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FOUNDED AND EDITED BY
HEINRICH STERN, M.D., LL.D.
New York



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Special Articles

THE SIGNIFICANCE OF THROMBO-PHLEBITIS IN
THROMBO-ANGIITIS OBLITERANS

By LEO BUERGER

Associate Attending Surgeon and Associate in Surgical Pathology, Mt.
Sinai Hospital; Visiting Surgeon, Har Moriah Hospital; Instructor
in Clinical Surgery, Columbia University
New York

There is no phenomenon of more importance in elucidating the true nature of the pathology of thrombo-angiitis obliterans than the characteristic thrombo-phlebitis or "migrating phlebitis" of this disease. The association of thrombosis of superficial veins of the upper and lower extremities with other evidences of obliteration of the larger arteries occurs in a sufficiently large number of cases to make the affection of the veins almost pathognomonic.

The name, *thrombo-angiitis obliterans*, was proposed by me in 1908 for that interesting group of cases of presenile gangrene which had been incorrectly described by the Germans under the name *endarteritis obliterans*. It was shown at that time that the pathology of the disease had been misunderstood, and that the lesions are the results of a thrombotic process, followed by organization and canalization of the occlusive clot.

In this paper we wish first to cite the histories of a sufficient number of cases of thrombo-angiitis obliterans with accompanying thrombo-phlebitis of the veins of the leg or arm to accentuate the

points by means of which a correct diagnosis can be made, and secondly, to discuss in brief the characteristic lesions of the veins, so that the importance of future researches on the etiology of the condition may be appreciated.

For the sake of clearness let us place the cases of thrombo-angiitis obliterans attended with thrombo-phlebitis or migrating phlebitis into five different groups. First, cases of thrombo-phlebitis without symptoms; second, thrombo-phlebitis with symptoms of limited vein involvement; third, migrating phlebitis causing the patient to seek treatment; fourth, cases in which both the migrating phlebitis and thrombo-angiitis obliterans play equally important rôles in the symptom-complex; and fifth, migrating phlebitis involving both the upper and lower extremities.

I. THROMBO-PHLEBITIS WITHOUT SYMPTOMS

There are patients who have no knowledge of the occurrence of any trouble in the veins of the leg, but in whose amputated limbs extensive old, or old and recent thrombo-phlebitis of the internal saphenous or its tributaries is discovered. Such a case was J. C., who could recall nothing referable to a disturbance in the superficial veins. Study of the vessels revealed old occlusion of a large part of the saphena by virtue of a thrombotic process, and some areas of more recent thrombo-phlebitis.

Case 1. J. C., 45 years, Russian Hebrew, admitted to Mt. Sinai Hospital May 18, 1908; has eight children (all well); gives a rather typical history of vascular disease of both lower extremities, resulting in amputation of the left leg at the knee. Four years ago he had "rheumatism" of the right leg with pain in the sole of the foot and redness of the toes lasting eight months. Since then it has not troubled him. The left leg, however, began to hurt him last summer; he could not walk a block without taking a rest. His big toe became "sore" recently, and now the pain in the foot is constant. He is told that the big toe is becoming gangrenous, and that his leg should be amputated, which he gladly permits.

With the observation just cited, no new clinical facts had been adduced, but certain similarities between the thrombotic lesions of the saphenous vein, as seen under the microscope, and the changes characteristic of the closed deep vessels were deemed sufficiently

suggestive to warrant the suspicion that here, in the superficial veins, a new territory for the process "thrombo-angiitis obliterans" had been found. We shall see in the histories that are to follow further evidence in favor of this assumption.

II. THROMBO-PHLEBITIS WITH SYMPTOMS OF LIMITED VEIN INVOLVEMENT

A more interesting group is represented in those patients who come to us with active thrombo-phlebitis and periphlebitic manifestations. Here and there along the course of the internal or external saphenous vein, alterations in the skin and subcutaneous tissues occur. These are in the form of small, erythematous, slightly indurated patches, about a centimeter in diameter, and tender to the touch. Were it not for the concomitant phenomena referable to the tributaries of the saphenous or the trunk itself, the nature of the appearance of these, however, or at other times in the course these cutaneous nodosities would have remained obscure. With the disease, cord-like thickenings of portions of the long saphenous, with or without adhesions to the skin, are frequently observed. As examples let us briefly tell the story of Cases 2 and 3 in Group II.

Case 2. S. S., 30 years old, Russian Hebrew, admitted to Mt. Sinai Hospital July 8, 1907; father of one child; has been suffering for four years with "weak legs"; for two years there has been pain in his left foot. About one and one-half years ago the second toe became gangrenous and was removed. Last winter his attention was directed to the blueness of the toes; it was difficult to keep the left foot warm. *For a couple of years he has noticed that "red spots" come and go along the inner and outer side of the shin bone.* They are a little painful and disappear without treatment. Now he seeks advice because the little toe looks as if it were going to die off. *Amputation* just above the middle of the leg.

Diagnosis.—A typical case of thrombo-angiitis obliterans with gangrene of the little toe of the left leg and cutaneous nodosities along the course of the internal saphenous vein from the ankle up to the region of the tubercle of the tibia; probably closure of a part of the saphenous vein.

The study of the vessels of the amputated leg showed extensive occlusion of the posterior tibial, anterior tibial, peroneal, and plantar

arteries (thrombo-angiitis obliterans). The long saphenous vein was filled for the most part with old organized tissue of a type indistinguishable from that seen in the deep vessels, and some of its tributaries were closed by more recent obturating masses. The cutaneous nodules corresponded to the distribution of the finer tributaries, but inasmuch as they had almost completely disappeared at the time of operation, no histological examinations were made.

As representative of the occurrence of migrating thrombo-phlebitis of the long saphenous and of erythematous nodosities in the same patient, let us cite Case 3, who observed and related quite accurately show the painful "hard cords" developed.

Case 3. F. S., 37 years old, Russian Hebrew, father of two healthy children, admitted to Mt. Sinai Hospital Dispensary April 13, 1909; says that he remembers having had peculiar pains in the soles of both feet on walking a few blocks for the last three or four years. About four months ago the big toe began to trouble him, but even before that he noticed *hard cords* along the inner side of the leg. Since then the nail of the big toe came off, leaving a raw wound which refuses to heal. The long, hard strands come and go; sometimes they are seen high up on the leg; at others, three or four inches above the ankle. *Besides this there are lumps further back on the inner side of the leg.* Patient does not return for treatment, so that the further course of the disease is unknown.

On physical examination the usual signs of thrombo-angiitis were found, with a trophic ulcer of the big toe. There were no evidences of recent thrombosis of the long saphenous other than one hard node four inches above the ankle; evidently the last attack of thrombo-phlebitis had subsided. The other leg showed somewhat less advanced symptoms of the disease.

Here, then, we are dealing with a case in which both the patient's narrative and ocular evidence point to the association of superficial and deep thromboses.

III. MIGRATING PHLEBITIS CAUSING THE PATIENT TO SEEK TREATMENT

When the attacks of migrating phlebitis make their appearance early in the history of the case, and when the attendant discomfort and pain are sufficiently great, then the symptoms belonging to the

true, deep-rooted affection—*thrombo-angiitis obliterans*—are sometimes wholly ignored by the patient and remain undiscovered by the physician. Medical advice is sought only for the “lumps” and “hard, tender strands” or “cords” that are oftentimes so disturbing. Such observations are of no mean importance in diagnosis, since they have taught me to seek for the early subjective and objective signs of thrombo-angiitis in every patient in whom there are spontaneous and unaccountable attacks of inflammation of superficial veins. Let us see what we can learn, then, from Group III, in which migrating phlebitis causes the patient to seek treatment.

Case 4. E. B., 36 years old, Austrian Hebrew, consulted me on January 17, 1909, with the history of having had stinging sensations on the inner side of the right leg, low down, some three months previously. A few days after the onset of this trouble he could feel a long, thickened “lump” behind the shin bone, a short distance above the ankle. Soon after this, another swelling, not unlike a “hard cord” appeared somewhat higher up on the leg, was very tender, and was succeeded not many days later by a third somewhat shorter strand.

Upon close questioning he admitted that although he seeks relief from the symptoms mentioned, he has been annoyed for almost a month before the beginning of the present affection by frequent *cramp-like pains* in the calf of the right leg upon walking a few (five or six) blocks.

Physical examination, January 17, 1909, revealed induration of the tissues about the saphenous vein, from the ankle to the upper fourth of the leg. The distal portion presents a cord-like thickening, with scarcely any inflammatory signs. Higher up, however, the skin is adherent to the deeper hardened area, and is exceedingly tender to the touch.

The dorsalis pedis and posterior tibial arteries of both legs are *pulseless*; the femorals and popliteals can easily be felt to beat. The big toe of the right foot has a cyanotic hue.

Course.—January 31, his phlebitis was found much improved; his right big toe often hurts him, and his foot easily gets “cold” and “tired.”

Two months later, March, 1909, no evidences of the old thrombo-phlebitis can be found. The big toe of the right foot still shows a

peculiar bluish discoloration, and the absence of pulsation in the vessels is the same as before. *There are no trophic disorders*; the most striking phenomenon is the vaso-motor disturbance in the big toe.

In short, we have here an exquisite example of a combination of early manifestations of thrombo-angiitis obliterans (pain on walking, evidences of disturbed circulation), with attacks of thrombophlebitis in the territory of one of the saphenous veins.

Whereas pathologic proof of the correctness of the diagnosis—thrombo-angiitis obliterans—is lacking in the last case, we are fortunate in being able to include here the history of another patient in whom there were similar symptoms, and in whose amputated limb and exsected veins we found ample material for anatomical investigation.

Case 5. M. K., 44 years, Russian Hebrew, father of three healthy children, was admitted to Mt. Sinai Hospital on December 8, 1908. His limbs never troubled him until about a year ago, when he felt the presence of tender spots on the inner side of the right foot. Soon other hard "lumps" and "cords" appeared; some of these in the neighborhood of the ankle, others higher up on the leg. After two months these disappeared, only to recur after a very short interval. Since then he has never been absolutely free from peculiar "painful spots," and now, on admission, he still has signs of some of them. About three months after the onset of these symptoms he experienced pain in the big toe, especially on walking. This has become gradually worse, so that he has been unable to get about properly for almost two months. Of late he has often had cramps in the calf and instep of the right leg after walking for a short distance. His chief complaint, however, is the painful condition of the inner side of his right leg.

Physical examination showed evidences of circulatory disturbance in the right lower extremity. Both the dorsalis pedis artery and the posterior tibial were pulseless, although pulsation of both the femoral and posterior tibial arteries could be easily detected.

Over the inner border of the right foot there is a red streak about one-half inch in length. This corresponds to a tender indurated mass which thins out and is lost as it is traced upward. A short distance below the middle of the leg the upper end of a hard cord

can be palpated. This extends down behind the border of the tibia for more than two inches, is adherent to the skin, somewhat nodulated, and marks the centre of an area of hypersensitive, swollen, turgid skin. There are no trophic disturbances. *Diagnosis*—*thrombo-angiitis, and thrombo-phlebitis of the internal saphenous and some of its tributaries.*

On December 15, 1908, a portion of the thrombosed saphenous was removed for pathological examination.

On December 26, 1908, the physical examination was recorded as follows: In the horizontal position the right foot has a light shade of red; this is most marked over the big toe, and fades off towards the ankle. In the web between the third and fourth toes there is a superficial ulcer. On the inner side of the foot, almost two inches from the internal malleolus, there is a hard, cord-like nodule which is adherent to the skin. Behind the tibia there is the scar left after removal of a portion of the saphenous vein. The saphenous can no longer be felt.

On elevation of the foot, blanching sets in rapidly and pain becomes intense. The pendent foot turns very red (marked erythromelia).

Further Course.—February 15, 1909, the pain in the foot has been getting steadily worse, and the fourth toe is beginning to turn black. On the 23d of February *amputation at the knee* was done, at the request of the patient, for early gangrene of the fourth toe.

What additional information did the autopsy of the amputated limb furnish? The prognostication that was made clinically in regard to the condition of the long saphenous vein was confirmed, for, as was expected, practically the whole of the main trunk of this vessel was found converted into a fibrous cord, the result of an old thrombotic lesion. It would lead me too far to describe the pathological changes, and I shall refer to these later on in a summary of what was characteristic of all the cases. Here let it suffice merely to mention that the obliterative process had much in common with that form which is typical of disease of the deep vessels. As for the arteries, the plantars, peroneal, posterior tibial, and lowermost portion of the popliteal were completely occluded by the brownish organized tissue usually encountered in the disease under consideration, whilst the deep veins were patent throughout.

For clearness, then, let us state our observation succinctly as follows: The case is one of *thrombo-angiitis obliterans*, in which the symptoms manifested themselves first in the form of migrating phlebitis that has persisted almost the whole of the year's course of the disease. The thrombotic lesion has affected the right leg and is associated with the development of typical symptoms of thrombo-angiitis. At the end of the year some of the deep vessels are closed, for there is absence of pulsation in the dorsalis pedis and posterior tibial. For a long time there are no trophic disturbances, but finally in the thirteenth and fourteenth months of the disease, ulcers develop and dry gangrene of one toe leads to amputation of the limb.

When the migrating phlebitis is a prodromal manifestation of the disease, thrombo-angiitis obliterans, or, if it marks a relapse in an apparently healed case, no phenomena referable to obliteration of the deep vessels may be obtainable. In such instances the excision of the affected superficial vein, followed by microscopic examination, will frequently reveal the typical pathognomonic lesions upon which a correct diagnosis may be based.

Case 6. H. P., 42 years, Russian Hebrew, seeks advice for a hard lump in back of the left leg on November 15, 1912. Twenty years ago the tip of the big toe of the right foot was removed in Russia, ostensibly for frost-bite. Since then (the exact date being unknown) the second toe of the same foot was also ablated. Save for these affections, no symptoms referable to the extremities can be recalled by the patient.

Physical examination shows a small thrombosed nodule, apparently associated with a varicose vein, over the calf of the left leg. In the vicinity there are small nodules, seemingly connected with tributaries of the external saphenous. There are no evidences of closure of the peripheral vessels.

Histological examination of the excised nodule, December 7, revealed the typical lesions of thrombo-angiitis obliterans.

Summary.—We have here, then, a case in which the history of the loss of two toes points to the existence of an old-standing thrombo-angiitis obliterans, the disease having become spontaneously cured. Recently there have developed evidences of involvement of superficial veins, the histological studies corroborating the diagnosis.

Another striking instance of the cases in which the thrombo-

angiitis obliterans symptoms are masked and not noticed by the patient, and where the patient seeks advice because of migrating phlebitis, is presented by the following case:

Case 7. J. W., Russian Hebrew, consulted me in November, 1911, because of red lumps in the left leg, and indefinite pains. He thinks he had syphilis 16 years ago, and that the lumps now present are due to this disease. The present trouble dates back about six weeks.

Physical examination shows a number of nodules of the usual type over the outer and posterior aspects of the left leg, and along the course of the internal saphenous vein.

On November 26, one of these nodules was removed for microscopic examination. Pathological examination shows the typical lesions of thrombo-angiitis obliterans in the early stages, typical giant cells, and miliary foci. Wassermann reaction on the 26th of November was negative.

December 4, some of the nodules had disappeared completely. The internal saphenous vein can be felt as a hard cord one-half way up the leg.

February 16, 1912, a fresh nodule has appeared above the Achilles tendon, another over the left calf, still another over the outer side of the leg above the external malleolus.

The dorsalis pedis and posterior tibial vessels of the right leg pulsate. The dorsalis pedis of the left does not pulsate. The posterior tibial pulsates very faintly.

Finally, we may cite as exemplifying cases of this group, the following history:

Case 8. B. C., 39 years, Russian Hebrew, seeks advice on account of pain in the left leg, which came on about four months ago. This seems to be associated with a nodule on the inner side of the middle of the tibia, and a similar nodule somewhat lower down. Three weeks ago another lump appeared on the outer aspect of the right leg. He has no pain on walking, and none of the symptoms of thrombo-angiitis obliterans.

Physical examination, December, 1911; several typical phlebotic nodules over the inner aspect of the left leg. The internal saphenous vein, from a point just above the ankle up to the upper fifth of the

leg, can be felt as a hard cord. Anteriorly, three inches above the ankle there are two fused nodules in a somewhat reddened skin.

The dorsalis pedis and posterior tibial arteries are not felt in the left leg. The posterior tibial pulse of the right leg is also imperceptible, though the dorsalis pedis pulsates faintly. There is no erythromelia, but moderate ischemia on elevation of both limbs.

In short we have here a case in which the symptoms of migrating phlebitis are prominent, the pulseless vessels and slight ischemia being the only evidences of thrombo-angiitis obliterans.

On December 5 one of the nodules was removed from the left leg for microscopic examination.

December 16. The phlebitis is extending from the region of the excised nodule in the upper part of the leg, and a distinctly tender cord, some $2\frac{1}{2}$ inches long, can be felt along the course of the saphenous.

December 22, 1911. The nodules in the right leg have almost disappeared.

Still more interesting and instructive are those cases in which the disease of the superficial vessels affects both legs and one or both thighs and in which the signs of

IV. BOTH MIGRATING PHLEBITIS AND THROMBO-ANGIITIS PLAY EQUALLY IMPORTANT RÔLES IN THE SYMPTOM-COMPLEX

Case 9 will illustrate this variety. The patient could be observed for almost a year, the progression of the obstructive changes in the deep vessels could be closely followed by proper interpretation of the varying circulatory phenomena in the leg, and many of the attacks of thrombo-phlebitis in the territory of at least one saphenous vein could be recorded.

Case 9. H. R., 32 years, Russian Hebrew, admitted to Mt. Sinai Dispensary August 9, 1908; has been suffering for five years. At first it was a burning sensation in the toes of the left foot that gave him most concern, but later on he was troubled more by his inability to walk distances on account of the sudden advent of attacks of pain that were felt from the toes upward almost to the knee. In cold weather he seems to be in poorest condition, for then his toes get cold and blue, and walking is very difficult. Although this has been going on for years, he has not found it necessary to consult

a physician until something else in his *right leg* began to engage his attention.

For the last five months long "streaks" or "swollen places" would come and go over the inner side of the right leg, behind the shin bone. These are often very painful. A week ago a physician told him that he had "phlebitis."

Physical examination on August 17, 1908. The vessels of the right leg pulsate, but the left posterior tibial and dorsalis pedis cannot be felt.

The right leg shows a tender cord with some edema around it, extending from the ankle almost to the tibial tubercle. This corresponds to the long saphenous. Erythromelia is definite on the left side; there are no trophic disturbances, and the circulation of the right leg is fairly good.

From now on aggravation of his subjective condition went hand in hand with the advancing lesions in the vessels. That an increase in the extent of vascular occlusion took place from this time on could be easily deduced from clinical observation.

On December 1, 1908, I have recorded the following: The right foot looks pale (evidence of the beginning of circulatory disturbances). After a short time it becomes slightly cyanotic. It looks cadaveric when raised for a short time. There is no erythromelia. The dorsalis pedis does not pulsate. A tributary of the long saphenous about two inches long can be palpated as a tender cord along the lower inner aspect of the right thigh; the skin over it is reddened. There are two nodosities in and under the skin below and to the inner side of the tubercle of the tibia. The left leg shows marked erythromelia; blanching in the elevated position is extreme; the popliteal is open, but the dorsalis pedis and posterior tibial arteries cannot be felt. There are no ulcers or other signs of trophic disorder.

The steady advance of the occlusive process in the deep vessels is well illustrated by the findings on December 1, 1908. In August all the vessels of the right lower extremity pulsated in normal fashion; now, in December, the dorsalis pedis is occluded. Corresponding with this there is a new symptom, the blanching of the foot. How remarkable that the disease of the deep vessels on the right side should be so closely associated with the attack of migrat-

ing phlebitis, the latter first attacking the saphenous in the leg, and now appearing in the thigh! We have evidences of chronicity in the affection of the superficial veins, and as regards the deep lesion, we have been able to watch its gradual development both by its effect on the palpable arteries and by the clinical manifestations it has produced.

On January 31 the big toe of the left foot was swollen and red; the nail was coming off. Immediately upon removing his shoe, the right foot had a very white color, but soon cyanotic patches mingled with the pallor all over the foot, especially in the region of the big toe. The pain in the left foot was now excruciating and he consented to an amputation with scarcely any reluctance. The left leg was amputated at the upper fourth.

Examination of the vessels of the amputated limb showed occlusion of the following arteries: dorsalis pedis, peroneal, plantars, and posterior tibial. The anterior tibial artery was open throughout most of its course. A large part of the long saphenous vein was found occluded by an organizing thrombotic process.

Diagnosis.—Thrombo-angiitis obliterans.

In short, our patient presents the following features of interest: (1) migrating thrombo-phlebitis of both saphenous veins; (2) involvement of the same vein in its course through the thigh; (3) associated progressive and synchronous development of the thrombosis in the superficial and deep vessels of the right lower extremity; and (4) absence of any cause for the lesion of the superficial vessels.

Case 10. W. T., 26 years, Russian Hebrew, admitted to the Mt. Sinai Hospital* July 10, 1909.

In April, 1907, he was treated for gangrene of the third toe of the left foot. Three months before admission to the hospital he had been suffering with pain in the left calf and foot. During the previous winter (1906) the left foot did not seem to be normal, so that he sought the advice of an orthopedist, who gave him the usual treatment for flat feet. Latterly, he has had severe pain in the calf, and shortly before admission gangrene of the third toe set

*I wish to acknowledge, with thanks to the members of the attending staff of Mt. Sinai Hospital, the privilege of studying the hospital material.

in. He had an amputation performed on the 27th of May, 1907, the left leg having been ablated at its upper third.

At that time my pathological studies revealed the usual changes that are seen with thrombo-angiitis obliterans. The dorsalis pedis, posterior tibial, the greater portion of the peroneal and plantar arteries were closed.

Present Status (1909).—Since discharge, June 27, 1907, until eight months ago, he seemed to be doing well. About this time (8 months ago) he noticed the appearance of red streaks and nodules on the inner side of the right thigh. After a few days these would disappear and new ones would appear in their stead, either higher up on the thigh, or near the knee. They caused a peculiar pricking sensation, and some were tender and painful. Lately, he has been able to walk no more than two hundred steps without resting.

The external manifestations on the 10th of July, 1909, were as follows:

A healed amputation scar in the left leg. In the dependent position there is marked erythromelia of the right leg. Neither the dorsalis pedis nor the posterior tibial can be felt to pulsate. On the inner side of the thigh, near its middle, there is a sensitive strand, which corresponds to the thrombosed saphenous vein. On the outer side there are a number of hard, indurated, reddened nodules. Over the inner side of the dorsum of the foot there are similar nodules and strands.

Diagnosis.—Migrating phlebitis and thrombo-angiitis obliterans.

In short, the history of this case reveals the following: Thrombo-angiitis obliterans first involving the left lower extremity, leading to amputation; insidious development of the same disease in the right lower extremity, with extensive thrombosis of the superficial veins of the thigh and leg.

The persistence of migrating phlebitis in the symptom-complex is illustrated by the following history:

Case 11. M. Gn., 28 years, Russian Hebrew, consulted me on the 13th of December, 1912. He has always been a heavy smoker (10-15 cigarettes a day), and began to smoke at 12 years of age. For about one year and a half he has had pain in both legs, particularly in the right, which prevented walking, causing him to take frequent

rests. He had been treated for about seven months for rheumatism in a dispensary, and also received mercurial injections.

Physical examination demonstrated a moderate degree of erythromelia of both feet, particularly of the left, and small typical nodules, evidencing involvement of superficial veins, over the territory of the internal saphenous of the left leg. Similar nodules were found in the right calf.

December 13, 1912, one of these nodules was removed for microscopic examination and studied in the pathological laboratory of the Mt. Sinai Hospital.

Neither the dorsalis pedis nor the posterior tibial arteries of either leg pulsate. Trophic disturbances are present in the form of a small ulcer.

Diagnosis.—Thrombo-angiitis obliterans involving both limbs with migrating phlebitis of both legs.

January 20, 1913, another superficial vein was excised and removed from the right leg for microscopic examination.

February 8, 1913, still another small vein was removed from the left leg for microscopic examination.

Patient was now lost sight of until June, 1914, when the left leg had to be amputated for gangrene.

June 16, 1914, my notes record that the left leg has been amputated; the right has distinct symptoms. There is chronic erythromelia, marked pain on walking, slight cyanosis in the dependent position, marked blanching on elevation.

June 24, 1914. He does not seem to be improved with the use of the diathermic treatment.

January 15, 1915. An ulcer has been present on the big toe of the right foot and spontaneously healed. The foot has improved considerably.

February 7, 1915. There are still tender nodules over the posterior aspect of the right leg. They are the typical nodosities belonging to migrating phlebitis. These have been present for six weeks. The right foot is atrophic, pigmented, the big toe fairly red; there is moderate erythromelia. *Patient complains only of the tender nodule in the back of the leg.* The left stump is in good condition.

Summary.—In short, we have here a case in which migrating

phlebitis was observed to be present off and on from December, 1912, up to February 12, 1915, recurring therefore more than two years and first observed in an acute or relapsing form; associated with it the usual involvement of both limbs, amputation of one limb having become necessary.

From the consideration of the data thus far presented it would appear that the internal saphenous vein is the site of predilection for that peculiar lesion which we have termed a migrating phlebitis. In July, 1904, I had the opportunity of studying my first case in which the veins of the upper extremity, too, were involved. Since then several additional patients with a similar distribution of the lesions have come under my observation. Four typical examples may be cited here.

V. MIGRATING PHLEBITIS OR THROMBO-PHLEBITIS INVOLVING BOTH UPPER AND LOWER EXTREMITIES

In three of the patients the disease has reached that stage of chronicity in which the suffering is almost constant and in which the limbs may be regarded as irretrievably lost. For there are cases that become "cured" as far as symptoms are concerned. And by "cured" in this sense we do not mean to imply that the pulseless dorsalis pedis, posterior tibial, or both, begin to beat again, but rather that, in spite of closed vessels, an adequate collateral circulation has become established, as evidenced both by the absence of the typical manifestations of impaired circulation, and by the patient's improved subjective state. These three patients *per contra* had the "severe" form of the disease, even though the issue, gangrene, was delayed far beyond our expectations.

Case 12. B. B., 34 years, Russian Hebrew, married, has no children; operator for eleven years. His malady began eight years ago, when he first experienced pain in the right calf on walking. He would be compelled to rest after walking four or five blocks. At about the same time he often noticed that there were long "hard cords" and "reddened lumps" over the front of *both forearms* (anteriorly) and over *both legs*. These would come and go, appear without provocation, now in an arm, now in a leg. The lumps were always small, pea-sized or slightly larger, and could be felt for two or three days.

He always felt better during the summer months. The nodules in the legs were present almost every winter for the first five years. Six years ago there was a "bad attack," in the course of which there were ("Adern") "veins" or "nodules" behind and above the right ankle. Then again, about three years ago, there was a repetition of this trouble. Nodosities formed behind the shin bone on the inner side of the right leg (region of saphenous) and the pain kept him abed for almost ten weeks.

Thus, up to this time he complained of the following: pain in the right calf on walking two to four blocks, painful nodules and cords, and cramps in the toes and sole of the right foot at night.

For two years the left leg has given him concern; the condition is practically the same as that of the right. Last winter, January, 1909, there were "sores"—one at the tip of the big toe of the left leg, and another at the end of the little toe of the right. He feels best when his legs hang down (a variation from the usual statement); but even in this position the toes often feel "dead." In the same way his fingers get "numb" in winter; he thinks that there is no blood in them.

Physical Examination.—In the right leg the toes have a tense, reddened appearance, the second and third being discolored most, the little toe having a cyanotic hue. Just behind the nail on the plantar surface there is a deep fissure, the tips of which are adherent. Slight pressure brings forth a drop of pus from the bottom of the wound. The erythromelia is marked over the dorsum of the foot, as well as over the sole. Ischemia in the elevated position is intense; this posture excites severe pain. The femoral artery pulsates; the popliteal, posterior tibial, and dorsalis pedis cannot be felt.

The left leg is similarly affected; the erythema is deeper and the toes are more swollen. There is a trophic ulcer at the tip of the big toe. The ischemia, too, is of a greater degree. All the vessels (femoral included) fail to pulsate.

Summary.—This is a case which, according to the story, combines thrombo-angiitis obliterans with migrating phlebitis of both upper and lower extremities. There are at present no evidences of involvement of superficial veins.

One of the most instructive of the cases of this series is a patient in whom the attacks of inflammation and thrombosis of superficial

veins dominated the clinical course for years before the symptoms characteristic of thrombo-angiitis obliterans came into evidence.

Case 13. D. B., 35 years, Russian Hebrew, first seen by me July 16, 1904, when at Mt. Sinai Hospital. He had been treated in the hospital eight years previously for "phlebitis" of the right leg; a portion (5 inches) of a large vein was diseased at that time, and the history states that the process was "migrating," moving up and down the thigh. He says that this trouble lasted off and on for two years. In 1903 there were "lumps" in and under the skin of the right leg, and then, three months later, in the left leg. Such swellings would last a week, develop into hard "tender spots" with a covering of red skin, and on one occasion three such spots appeared on the left arm, in front of and just below the elbow.

Physical examination, July, 1904. In the left antecubital region there is a thickened, slightly reddened cord about two inches long. Another is situated on the ulnar aspect of the same forearm, near the elbow. The right forearm presents a similar vein about three inches from the elbow; the skin is not reddened. On the inner side of the right cubital space a subcutaneous adherent nodule can be felt; it is very tender. There are several such nodules in the right calf and smaller ones over the left shin bone. No edema, but slight cyanosis of both legs in the pendent position. A portion of one of the thrombosed arm veins was extirpated for study.

Course.—A year later, 1905, symptoms referable to affection of the deep vessels of the left lower extremity manifested themselves, to wit: coldness and blueness of the left foot and superficial ulcers on the toes.

Thus far our patient presented no striking addition to the symptom-complex under discussion, other than the thrombo-phlebitis of the arm veins. In 1907, however, he developed a gangrenous patch at the tip of the middle finger of the right hand. This rather unique site for trophic manifestations is rarely seen in obliterating thrombo-angiitis, and therefore deserves more detailed mention.

On February 1, 1907, D. B. consulted me at the Good Samaritan Dispensary. His doctor had been treating him for a "felon" of the middle finger of the right hand. His hand had been cold for several weeks, and the middle finger was painful. Four weeks previously a black "dead" spot formed on the tip of the finger, and

since then, what with cutting it and self-treatment, he thought that the present intensely painful affection had overtaken him.

Physical examination, February 1, 1907. A portion of the tip of the middle finger is gangrenous; there is no infection; the distal phalanx seems to take part in the process of mortification. On the dorsum of the hand, just over one of the veins, there is a bean-sized indurated area; the skin over it is adherent and tender. About one inch above the wrist, behind the radius, there is a reddened hard cord, more than an inch in length (doubtless a thrombosed vein).

The left foot is bluish, and there are a number of red nodosities in the leg. They are placed over the course of the long saphenous vein, one or two inches above the tip of the malleolus, and a couple of others three to four inches above the ankle. The right leg shows a thrombo-phlebitic, indurated process over the lower part of the anterior tibial group of muscles.

Further Course.—The finger improves very slowly; in April it is healed. The nodules in the upper extremities disappear after three weeks. April 16, 1907, over the outer side of the right leg, four inches below the tibial tubercle, the skin and subcutaneous tissues are indurated. There are two hard areas further down. The nodosities come and go, now in the right and now in the left leg. On April 29, 1907, his left foot troubles him greatly. It is slightly swollen; the toes become deep red in the dependent position. The right foot is slightly red in the same position. The femorals and popliteals pulsate well. On June 1 the left foot is very painful; the toes feel as if needles were sticking them.

November, 1907. Since the beginning of September the right leg seems to be affected by the same disease as the left. New nodules of subcutaneous infiltration have appeared on the inner side of the left leg and the inner side of the right knee. They seem to have very little tendency to disappear. He often has pain in the middle finger of the right hand, and this hand is colder than the left.

Physical Examination.—On holding both hands above his head the right becomes blanched. When the hands hang, the right becomes cyanosed; there is an admixture of red, so that there is a mottling of red and blue (erythromelia of the upper extremity). Both radial pulses are good. *Lower Extremities*.—On the inner aspect of the right leg three nodules are seen, two near the tubercle

of the tibia, a third one inch behind the middle of the crest of the tibia. They are $\frac{3}{4}$ to 1 inch in diameter, involve skin and subcutaneous tissues, and are red. Similar infiltrations are found on the inner side of the left leg, two in the middle and upper third; two others four inches above the ankle. They evidently follow the course of the saphenous vein. The right foot has a bluish-red color. The left is even more markedly discolored; the second toe is enlarged, looks angry, and presents a small superficial ulcer near the nail. On December 19 (a warm day) the legs are red when they hang down. There is no cyanosis. Both feet become cadaveric when raised. A new cord has formed over the right wrist; it is about an inch long, and lies over the radius; a somewhat longer cord is situated over the inner side of the right knee. (Salicylate of mercury injections are administered.) On January 3, 1908, the dorsalis pedis and posterior tibial arteries are pulseless. On February 7 he still has the painful cord over the right wrist, although he has had seven injections of mercury. The right popliteal pulsates, the left pulsates faintly; both femorals are open.

September 7, his right foot is worse than the left. The dorsalis pedis and posterior tibials of both legs are evidently closed. At this time the right popliteal does not pulsate; the left beats faintly (note that this corresponds with the aggravated subjective sensations of the leg); both femorals are felt. Recent ulceration has occurred in the web between big and second toes of the right foot. The toes are intensely red in the dependent position. On November 19, in the horizontal position, both feet possess a marked erythematous hue.

On April 13, 1909, the patient came to the hospital for the ulcerated condition of both feet; he cannot walk. Over the dorsum of both feet there are superficial ulcerations, and there are a number of trophic ulcers in the webs of several of the toes. Under rest in bed and local treatment all the wounds heal. By June 9 both legs are in a condition of chronic erythromelia, even in the horizontal position. The feet have a dusky red hue; in the dependent position there is an admixture of purple. The skin is shiny and appears thinned, although the toes themselves are enlarged. Only the femoral arteries pulsate; the ischemia in the elevated position is very marked; all the superficial ulcers have healed.

Summary.—The total history up to the present time extends through a period of about twelve years. During the first eight years the clinical course was characterized by repeated attacks of migrating phlebitis of the superficial veins of the upper and lower extremities, and the appearance of cutaneous nodosities, due in all probability to circumscribed venous thromboses. These attacks were accompanied by the usual pain and tenderness, some edema, and secondary cutaneous manifestations. Towards the end of the first period the prodromal indefinite pains of typical thrombo-angiitis were noticed. These were followed by the development of marked erythromelia of the left lower extremity, and of trophic disturbance. Then came a cessation of the process on that side, only to give way to a similar diseased condition on the right side, where it has caused obliteration of the distal vessels and the popliteal. In short, a period of occlusion of superficial veins was followed by a period of arterial occlusion which attacked first the left, and then the right leg.

Does the paroxysmal nature of the involvement of the superficial veins throw any light on the sequence of events in the deep vessels? From my previous pathological studies* it seemed most plausible to assume that certain territories of either arteries or veins become rather suddenly thrombosed, after a fashion similar to the thrombotic process occurring in the superficial veins of the lower extremities. The history of the fourteenth case is exceedingly illuminating on this point, since it suggests that attacks of migrating phlebitis of one leg may occur synchronously with paroxysmal pains in the other leg, and that these latter pains are closely associated with other signs clearly pointing to an exacerbation of the thrombotic lesion in the deep vessels—"an attack (if we may so regard it) of thrombo-angiitis obliterans." In other words, it seems more than likely that, at any given time, the patient may be suffering from a more or less acute disturbance, in the course of which both superficial and deep vessels become closed.

Case 14. M. P., 34 years, Russian Hebrew, admitted to Mt. Sinai Hospital in May, 1908. My history was taken on May 24, 1908. Two years ago there were some "swollen places" on both legs, and he had pain in the legs when he walked. One year ago he had attacks of "phlebitis"; this was the diagnosis at the Presbyterian Hos-

*Am. Jour. Med. Sci., October, 1908.

pital. The veins on the inner side of the left forearm and arm, almost up to the armpit, were painful. The left saphenous at the middle of the leg was also diseased at that time. He had been treated at Mt. Sinai Hospital in August, 1907 (service of Dr. Manges) for "phlebitis migrans." At that time no suspicion was entertained as to the existence of the condition, thrombo-angiitis obliterans.

Last winter he often had pain in the feet on walking, and this has been much worse for the past four weeks. During the last two months the symptoms of phlebitis have recurred in the left leg and the left arm.

Present History.—For four days he has had excruciating pains in the calf of the right leg, even when in bed. Besides this, he has painful cords and "spots" in his left leg.

Physical Examination.—Both radials pulsate. The patient seems to be very restless because of the pain in his right leg. In the right leg neither the dorsalis pedis nor the posterior tibial artery can be felt; the popliteal artery is patent. The toes are slightly red in the horizontal position; there is marked erythema of the toes in the pendent position. Ulcers and thromboses are absent. (Note made May 24: The pain in this leg must be interpreted as suggesting thrombosis of the deep vessels, because there is nothing else to account for his suffering; apparently no neuritis.)

In the left leg also absence of pulsation in the dorsalis pedis artery and posterior tibial artery is noted. Just behind the tibia, at the middle of the leg, the saphenous vein is thrombosed, being adherent to the skin, which is reddened. There are a number of nodules in its vicinity, probably corresponding to small tributaries. There is erythromelia of moderate degree, but no marked ischemia in the elevation posture. The popliteal artery is patent.

In the left arm a small portion of an anterior ulnar vein, low down, is indurated.

Briefly, then, the typical signs of bilateral thrombo-angiitis obliterans, without trophic disturbances, varicose veins, or infection, are associated with attacks of thrombo-phlebitis of the superficial veins of the upper and lower extremities.

On May 28, 1908, the pain in the right leg is gone, the cords are disappearing, the ulnar thrombosis is no longer palpable.

I saw the patient again on December 1, 1908. After leaving the hospital he could walk but a block without stopping for a rest. For about two weeks a new longer cord has traveled up from the middle of the inner side of the left leg, behind the knee, to the lower part of the thigh. There is another one behind the ankle and inner side of the foot. In the calf there are two tender bean-sized nodosities. He says that the big toes always feel as if they were asleep, and he often has an inclination to rub them to dissipate the feeling of numbness. Examination shows his condition to be slightly worse than it was in May, as regards sufficiency of circulation in both legs.

On June 15, 1909, he was again examined by me. He was then a pitiful spectacle to behold. Pulling himself along on two crutches, with an expression of fear written all over his face lest the contact of the soles of his feet against the ground call forth excruciating pain, with the aid of his wife he finally seats himself, telling me the following story: He has tried "everything" for his legs. He has been treated in other hospitals since I last saw him, and now he cannot walk at all. The big toe of the left foot hurts him unbearably, and his physicians are unable to ward off the coming of those dreadfully painful "sores" and "fissures" that form without reason on his soles, between the toes, and near the borders of the nails. He cannot bear his weight on the legs at all. The effort to walk was soon given up and he has permanently assumed the horizontal position as the only one possible to be borne..

Physical examination shows intense erythromelia of both feet, with a slight cyanotic hue, as in Case 12, D. B. The middle portion of the internal saphenous vein for about an inch of its course through the leg is converted into a hard, tender cord. There is a nodule 0.5×1 cm. three inches below the left tibial tubercle and two inches outside of the crest of the tibia. All the toes of both feet are somewhat enlarged; they look stiff and turgid when held in the dependent position. The blanching of the raised feet is extreme.

Resumé of Case 14.—Recurring thrombo-phlebitis migrans of both upper and lower extremities, gradual development of the severe chronic clinical type of thrombo-angiitis obliterans without gangrene, symptoms indicating the simultaneous paroxysmal attack of superficial and deep vascular channels.

One of the best examples of extensive disease of the veins in all four extremities is that presented by Case 15. Because of the development of an adequate collateral circulation, obstruction and closure of the deep vessels had apparently produced no symptoms in the left leg, whilst the disease had made considerable progress in the right leg. Signs of an active migrating phlebitis could be found only in the lower extremities, but the definite statements of the patient leave no doubt as to the correctness of the view that he had had attacks of phlebitis in the upper extremities at one time.

Case 15. M. G., 37 years, Russian Hebrew, married, has two healthy children; consulted me August 6, 1909. Four years ago he had tender "cords" or "lumps" on both forearms and also on the inner side of the arms. These soon disappeared and have not recurred; at that time, however, there was also a similar condition in the calf of the right leg and inner side of the left leg. He was quite free from trouble until a year ago, when similar painful spots developed in the legs.

For three months he has had wakeful nights, because of pain in both feet, especially in the right. His toes get cold and he cannot walk because of the sudden advent of cramps in the calves. The "cords" in the right leg have now disappeared, but they are still present in the left leg, where they come and go.

Physical examination, August 6, 1909. Both lower extremities present the typical signs of thrombo-angiitis obliterans. Evidences of circulatory insufficiency are most marked in the right leg, where there is marked erythema in the horizontal and pendent positions. Both legs become intensely blanched when elevated. All three vessels (dorsalis pedis, posterior tibial, and popliteal) are pulseless on the right, whereas a very faint pulsation in the upper part of the left popliteal can be detected, the distal vessels evidently being closed. There are no ulcers.

Just below the middle of the course of the saphenous of the left leg a hard, knobbed, tender cord can be easily felt. At the inner border of the foot there is an erythematous nodule which is tender.

On August 8 about one inch of the thrombosed saphenous of the left leg was excised under local anesthesia for diagnostic purposes. The vein was filled with recent clot, and was fairly adherent to its bed, showing an active periphlebitis.

In September, 1909, the right leg was amputated three inches below the knee by Dr. Hurley of the Sydenham Hospital, because of gangrene of the toes.

On October 27, 1910, he says that there was considerable pain in the sole of the left foot, and that he could not walk more than two blocks without stopping for rest. Ever since the operation he has had recurrent attacks during which the same hard cords or nodules which he had before developed in the left leg. Several of these are now present, and he has had them for three weeks.

Physical examination, October 27, 1910, shows a nodosity in the middle of the left leg, and two or three confluent nodosities above the malleolus. There is marked erythromelia, and the pulses are absent in the popliteal, posterior tibial, and dorsalis pedis.

November 10, 1911. The phlebitic process is still present. Fresh nodules are making their appearance. The evidences of obliteration of deep vessels of the left leg are more striking, and the disease, thrombo-angiitis obliterans, is evidently making progress, with signs of the development of trophic disorders.

Epicrisis.—We are dealing here with a case of bilateral thrombo-angiitis obliterans with associated migrating phlebitis which had originally affected both forearms, and for more than two years has shown itself also in the lower extremity. The persistence of the migrating phlebitis and the chronicity of the deep vessels, thrombo-angiitis obliterans, of the left leg, are features worthy of note.

Here (Case 15) we are dealing with a case presenting active signs of thrombo-angiitis of the vessels of the lower extremities for three months. At the same time, there were recurring attacks of phlebitis of the upper and lower extremities. At times he sought advice because of the phlebitis; at other times, because of the symptoms referable to the deep-seated disease. Four years after the onset of the disease the findings were as follows:

The results of advanced closure of the arteries of both lower extremities; absence of any recent or active symptoms in one of the legs; distinct signs of a slowly progressing involvement of the circulation of the other limb, with recurring attacks of phlebitis of both saphenous veins, without ulcers, trophic disturbances, varicosities, or evidences of inflammation.

These facts lead us to the following conclusions:

First, that the phlebitis plays no subsidiary rôle in the symptom-complex of some of these cases.

Second, that the disease, when it affects the upper extremities, is less enduring than in the lower extremities.

Third, that we have here another link in the chain of evidence speaking for an identical cause for the disease of the deep arteries and veins and the superficial veins.

PATHOLOGY

In 1908 our studies of the pathology of nineteen amputated limbs in thrombo-angiitis obliterans had clearly demonstrated the thrombotic nature of the vascular occlusion. It was also shown that the pictures formerly interpreted as results of a thickening of the intima were produced by organization and canalization of red obliterating thrombi. It was found that the disease involves the deep arteries and veins of both the lower and upper extremities, commencing by preference in the vessels of the foot, such as the dorsalis pedis and plantars and their larger branches, ascending so as to sometimes close even the iliacs and aorta. Clinical and pathological data led to the assumption that the progression of the thrombotic process takes place rather in attacks or sudden exacerbations than by a gradual ascent; that larger or smaller territories of the deep vessels become suddenly closed, just as the saphenous veins are wont to be thrombosed and inflamed from other causes—in other words, that the process is a migrating thrombosis of the deep vessels comparable to the migrating phlebitis of the extremities.

A cursory study would lead one astray as to the significance of the most common lesions seen in the arteries and veins, for it would fail to reveal the fact that there are two distinct phases in the pathology of the disease. The lesion most commonly encountered is but the result of the organization of thrombi, and of importance in our investigation only in so far as it is productive of the pictures that may be confused with endarteritis obliterans. More interesting and more valuable for investigation is the "acute stage," or earliest lesion, that occurs simultaneously with, or shortly after the onset of the thrombosis. This early stage was found in the vessels of but two of the amputated limbs. In these certain specific morphological alterations were encountered, whose meaning was

not understood at that time. These histological changes appeared to be characteristic of the disease, thrombo-angiitis obliterans, not having been met with in vessels thrombosed through other causes. The regularity of the occurrence of the typical lesions aroused the suspicion that here was a specific morphological alteration, due to a specific cause.

In short, whereas the usual changes in most of the vessels of an amputated limb represent the healed stage of the disease, that in which a fibrous mass containing canalizing vessels has taken the place of the original clot, there is another early or acute stage of the disease which alone is of value in throwing light upon the true nature of the process. It is only at this particular period in the history of the pathological process that the media is diffusely infiltrated with leukocytes, and that the lumen is filled with red clot, in which certain typical miliary giant-cell foci* make their appearance. It is these foci that lend a characteristic appearance to the thrombotic lesion of thrombo-angiitis obliterans.

When these lesions were first referred to in 1908 their significance was not understood, although the suspicion was already aroused at that time that they were specific for the disease and probably represented a peculiar reaction on the part of the tissues to some toxin or organism. It seemed clear, too, that it would be a difficult matter to obtain an adequate amount of material from the deep vessels for the study of the acute stage of the disease. It was here that we had to pause in our deductions, when we were fortunate enough to encounter a most interesting fact, that the superficial or subcutaneous veins of the upper and lower extremities may also be affected by the disease, thrombo-angiitis obliterans. Thus, in 1909, the association of migrating phlebitis of the subcutaneous veins of the extremities was noted in eleven cases. From a study of the clinical history of the cases, and of the histology of the affected subcutaneous veins, exsected during various stages of the disease, the following conclusions were drawn:

1. The disease thrombo-angiitis obliterans is often associated with thrombo-phlebitis of superficial veins of the arms and legs.

*Buerger, *Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie*.
21 Band, 1910.

Do—*Am. Jour. Med. Sci.*, October, 1908.

2. Certain peculiar cutaneous nodosities are characteristic manifestations in many cases.

3. The disease of the superficial veins may be subsidiary or it may dominate the clinical picture. Objective signs referable to these vessels should be regarded as extremely suspicious marks of the synchronous development of thrombo-angiitis obliterans, in the form of pulseless vessels, erythromelia, blanching of the leg in elevated posture, cold and blue toes, pain in the calf of the leg brought on by walking, and other typical phenomena.

4. Migrating thrombo-phlebitis may give no symptoms, the signs referable to the deep vessels being of most importance.

5. Patients may suffer at one time from migrating thrombo-phlebitis, at another from the progress of the occlusive change in the deeper vessels.

6. One of the cases suggests the possibility that attacks of trouble in surface veins may occur simultaneously with similar exacerbations of disease in deep vessels of another limb.

7. The morbid process resulting in the production of cutaneous nodosities and thrombosed superficial veins is independent of varicosities, of infection, or of trophic disorders in the territory which they drain.

8. The vessels of the upper extremity may be affected by the lesion thrombo-angiitis obliterans.

9. Thrombo-phlebitis in the arm and forearm should arouse suspicion as regards involvement of the deep vessels of the legs.

10. Further studies should be directed towards solving the relationship between the two thrombotic lesions that we have described. Perhaps excision of nodules and veins early in the disease, exploratory incision for inquiry into the condition of the deep vessels, and bacteriological and serum investigations along the proper lines will do much to enlighten us in our interpretation of this most puzzling symptom-complex. Although absolute proof is lacking, it seems more than probable that the same determining causative factor is responsible for the lesions of both the superficial and deep vessels.

Since 1909 I have been able to gather data on fifteen additional cases in which the superficial veins were involved and have brought the number of excised veins up to twenty-five. In these, both the acute and healed stages of the disease were found. From a con-

sideration of the pathological pictures the conclusion was reached that the specific characteristic lesion of thrombo-angiitis obliterans may affect the deep as well as the superficial vessels; that it is in the veins that we shall have to look to find material for investigation of the causative agent; and that not only do the superficial veins present the typical military giant-cell foci, but they also demonstrate that these foci are a later stage, or attempt at organization of purulent foci. In other words, the finding of miliary pus foci in the subcutaneous veins as precursors of the typical giant-cell foci was noted in a sufficient number of instances to warrant the conclusion that this lesion represents the acute stage of the disease, and suggested, too, that the thrombotic process must be caused by the presence of some organism.

From the investigation on the twenty-five exsected superficial veins, with a comparative study of the lesions in the deep vessels, the following conclusions were drawn:

SUMMARY

An acute inflammatory stage is the initial manifestation of the pathology of the disease, thrombo-angiitis obliterans. Certain purulent foci are developed strongly suggesting the presence of some specific toxin, or, more probably, some microbial agent. The destructive influence of these foci is evidenced in their action on the angioblasts, whose true purpose is thwarted, the result being the elaboration of a morphologic complex, distinctive and characteristic for the disease. Just as in tuberculosis, and doubtless in Hodgkin's disease, certain well-known structural products represent the specific reaction of the tissues to some organism, so here, too, the changes described are absolutely typical and diagnostic. Their *raison d'être*, it is hoped, has been explained.

Besides these changes, the production of aseptic, bland clot plays a part in the development of the occlusive tissue; whilst the infiltration of the media and adventitia makes for the adhesive fibrosis in the deep vessels, and the distinctly palpable products of periphlebitis in the subcutaneous veins. The change of the acute into the healed connective stage is rapid, a type of occlusion resulting which has for a long time been regarded as due to an "endarteritis obliterans."

If we have been unsuccessful in our search for the offending agent, it is hoped that we have at least clearly shown how the problem should be attacked, and where we must expect to find the causative agent of the disease.

Certain it is, then, that the recognition of the associated migrating phlebitis is not only important in diagnosis, but affords the terrain for the discovery of the etiology of the affection.

THE DIAGNOSIS OF THE BORDERLAND PSYCHOSES: A WARNING

By L. HARRISON METTLER

Professor and Head, Department of Neurology and Clinical Neurology,
College of Medicine of the University of Illinois; Neurologist,
Norwegian Lutheran Deaconess Home and Hospital
Chicago

"We must not forget that, as the doctor shows, and must show, a searching interest for the psychology of his patient, so, too, the patient, if he has an active mind, gains some familiarity with the psychology of the doctor, and assumes a corresponding attitude toward him. . . . Therefore, I maintain that a doctor must be analyzed before he practices analysis. . . . The Indians, when America was discovered by the Spaniards, took the horses of the conquerors, which were strange to them, for large pigs, because only pigs were familiar to their experience. This is the mental process which we always employ in recognizing unknown things."—JUNG.

The term psychosis has a broader meaning than that usually given it by the psychiatrists. It is a stock word in psychology and means merely a "change in the field of consciousness," or better still, "the mental constitution or condition." It is in this broader sense that the psychoses will be discussed in this paper. Again, it will be noted that I have used the adjective *borderland*, instead of "*borderline*" so common among the authors, in the title of this paper. There is no "*borderline*" among the psychoses, for human mentalization is a graded manifestation, from idiocy at its lowest point to an indefinite, ever-advancing high point. There is no fixed point

or middle line anywhere in this concept of universal mentalization. The normal for any one time, place, or race has not, and probably never will be established. The nearest approach to a standard of measurement possible is that of a *numerical average*. The average, however, is always a shifting and variable factor. The average of one nation is not the same as that of another. The average of one age is not the same as that of another age. The average mentalization in Africa is not the same as that in Europe and neither can be called in the true sense the normal. The average mentalization of the ancient Greeks is not the same as that of the modern Greeks, nor can either be strictly referred to as the normal. Psychosis is not a thing or entity; it is merely a reaction, an appearance, an outward manifestation, complex and variable, dependent upon innumerable underlying factors concerned with past influences (heredity, organic evolution, etc.) and with present influences (physiological function, environment, etc.). This relativity, variability, and instability being true of the average psychosis, it is obvious that no strict definition of the abnormal psychosis, or insanity so-called, is possible. Only well out at the extremes of this graded universal mentalization can a clear picture be drawn as to what for convenience may be spoken of as insanity. No mind at all or idiocy and gross imbecility can be easily recognized. Unusual mentalization or exaggerated degrees of psychosis can be clearly detected. Neither idiocy nor genius being within that broad midland of averages, both are put down as forms of insanity, as equally abnormal psychoses, freaks of nature. If our definitions, classifications and diagnoses of insanity could be limited to these gross pictures of mental aberration we might more often find ourselves in the right. At least we should not be subjected to such witticisms as that all the world is insane but part of it builds asylums to incarcerate the other part in order to make itself feel that it is not quite so insane. It is because of our inability to define both great wit and little wit that the poet is justified in exclaiming, "Great wits are sure to madness near allied and thin partitions do their bounds divide," and the philosopher in writing, "The insane, for the most part, reason correctly but from false principles; while they do not perceive that their premises are incorrect." According to the former, many an inmate of an asylum might well have been given a niche in the Hall of

Fame; while according to the latter, many a supposedly sane individual would be occupying a cell in the State institution. So-called insanity is a gross affair and being always the expression of some bodily or organic ailment or deficiency, is not hard to diagnose when an exhaustive examination of the patient is made. Now, most of the writers upon the so-called borderland cases that I have read speak of the frankly defective and grossly abnormally acting individuals. In a word, their borderland, or as too many of them unfortunately say, "borderline," cases are merely definite, mild degrees of permanent mental aberration. These constitute a large and most interesting class. Today they are being studied more than any other group of cases. Heretofore they have been granted too much liberty and as a result society has often become the victim of their crimes and grossly anti-social ideas. This whole class of *defectives*, easily recognized when thoroughly studied, and calling for better and more radical control than has heretofore been given them, is not what I mean by the borderland psychoses. The cases I refer to are *actual borderland* cases. Some of them are on one side of the borderland and some of them on the other. The same case may at one time be regarded as insane, at another as perfectly sane. These doubtful cases that flit, as it were, through the hazy area between health and disease are the ones that give us our real difficulties in diagnosis. Physically and organically, so far as all outward signs indicate, they are healthy. Their physical examination usually reveals nothing abnormal. Their heredity may be negative and their environment seems to be all that could be desired. And yet they think and act queerly at times and not infrequently have been sent to the psychopathic hospital. How are we to detect these cases? Are there any reliable criteria by which we can pronounce them sane or insane? What is sanity or insanity as applied to them? They certainly do not think and live like their milieu. They even cause much anxiety and disturbance. How insane, or how likely to become insane, is a psychoneurotic of this sort? These are a few of the questions which we all devoutly wish at times that we could answer. These are the *real borderland* cases that tax our skill and patience to the limit.

One thing is fully recognized today in psychiatry, namely, that it is not the mind so-called that is diseased but the body or organ of

whose function the mind is but the expression. In other words, *insanity always means organic disease or inadequacy*. A psychiatric diagnosis always depends for its accuracy upon the diagnosis of the underlying functional and organic abnormality. This is not always easy because the inherited weakness, the degenerative inadequacy, the acquired toxemia, the parasitical infection or the pathological tissue change may all be so slight or beyond our means of research as to be quite overlooked. In such a case the diagnosis of the mental malady, no matter how well the mental symptomatology presents itself, lacks more or less in completeness and definiteness. An element of error becomes more and more insistent in proportion to the absence of this underlying organic diagnosis. This is the reason why errors are so common in the diagnosis of mental diseases—errors which are obviously and manifoldly enhanced by the want of thoroughness on the part of the ignorant or careless examiner. With care and thoroughness, resulting in the detection of some basic organic disease, a fairly positive diagnosis of the nature of the mental aberration may be established when the latter is of the slightest and most evanescent character. Like a “pathognomonic symptom,” it may be said, the presence or absence of the underlying organic disease fixes the sane or insane character of the victim’s mental vagaries. The only question at issue is the discovery of this underlying disease. Here we approach the borderland type of psychoses as I understand the term.

There are cases, however, calling for a psychiatric diagnosis in which the most careful and complete examination fails to reveal any organic basic disease. These may be cases of incipient disease, as, for instance, general paresis in which for some reason or other the tests for luetic infection have all proved to be negative (and every one of large experience has seen a few such cases); or they may be cases whose mental manifestations may be due to strange and unusual causes not falling under the general head of pathology. In both sets of cases time usually reveals the real nature and cause of the apparently unusual psychosis. Hence the frequent necessity of a preliminary observation and treatment of these cases in a sanitarium before a positive diagnosis can be established.

There are times when it is desirable, however, to make a diagnosis, if at all possible, without delay. At all events, the laity

usually insist upon some sort of a diagnosis and prognosis before waiting so long. In these cases where all physical findings are apparently wanting and yet the patient seems to act queerly and to be quite out of harmony with his immediate and general mental environment, the examiner is forced to make some sort of a diagnosis upon the *purely psychological data* presented by the one being examined. The question becomes one then of *psychology*. This sort of an examination is unique. There is nothing like it in the entire realm of medicine and surgery. It is one mind examining another mind; like a definition trying to define itself; a use of terms used to explain themselves. Blunders and disasters frequently follow efforts in this field; so much so that many physicians wisely refuse to pass any opinion, while many more unfortunately illustrate the saying that fools rush in where angels fear to tread. The examiner's own psychosis is here as much at stake as that of the one being examined.

In these cases all the psychical exhibitions may be, or seem to be, aberrant, but usually the trouble lies markedly in one sphere alone. The memory, the imagination, the volition, the reason, the attention, the consciousness, the final judgment may all be out of harmony with the requirements of the environment; but as a rule the trouble will be found to be chiefly in the realm of the *ideas*, with all that the word idea connotes.

Tanzi writes that "irregularities of ideation form one of the two foundations of clinical psychiatry, and indeed, the more characteristic of the two: they can be grouped into those that relate to the rapidity of association, the logical value of the ideas, their absolute quantity, and their arrangement." But just here is where the difficulty arises. Who and what is to establish the *proper rapidity* of the association of these ideas, or their *logical value*, the *proper quantity* that they should show, and *how they ought to be arranged*? The opinions of a Shakespeare or a Goethe in regard to all of these qualifications of ideas, if put into force, would send a good many supposedly normal people to the idiot wards of the hospital.

Here is just where our trouble begins in the diagnosis of the real borderland cases wherein no discoverable organic lesion or intoxication can be established. *The symptomatology of these cases is wholly of the psyche and the presentations upon which we must*

depend lie wholly in the sphere of the ideas. It is because of this that craziness has been satirically declared at times to be universal; that discoverers, inventors, writers, artists, scientists, reformers, and enthusiasts of all sorts are sometimes seriously regarded as mentally unbalanced; that genius has been identified with insanity by so many of the followers of Lombroso; that faddists, ritualists, followers of all sorts of isms, Christian Scientists, spiritualists, and other religious and semi-religious devotees have been regarded at times as more or less crack-brained. It is a human weakness, a phase, as it were, of human objectification, shown individually and racially, to feel and say, "We are sane; all others whose *ideas* do not agree with ours must be somewhat insane." This is the popular logic, and as such can well be allowed to pass; but unfortunately the same sort of logic has too often invaded scientific circles, and today, in some quarters, seems to be dominating our psychiatry. I refer, of course, to some of the conclusions that emanate from the Freudian school of psychoanalysis, from the extreme believers in the value of such mental tests as the Binet-Simon, Yerkes, and others, and from a few of the teachers and writers of psychiatry who over-value the classification of mental symptoms, calling them mental diseases.

In legal phraseology a man should always be tried by his peers. The same general principle should obtain when one mind, or set of minds, examines another mind. When an individual is regarded as queer and is presented for examination, the whole realm of his ideas should be taken into consideration and the force and value of those ideas met by a similar rapidity, quantity, and versatility of ideas on the part of the examiner or examiners. For example, I once saw in consultation a well-known concert violinist who was thought to be a very superior musical genius and was suspected of insanity only after he had smashed his violin and threatened the life of his mother. A long conversation with him in one of his lucid intervals upon music, of which I have made some study, showed me that in this art alone an earlier examination would have demonstrated that not only was he most superficial in his knowledge and conception of this, the greatest of the arts, but his wonderful rendition of it was more or less akin to that of Blind Tom or of what the

French would call the idiot-savant. This patient, instead of being a great artist, was a defective and has long been in an asylum.

Per contra, let me cite the following illustrations, all of whom were presented as cases of possibly serious mental alienation.

A very successful business man was depressed and melancholy, even to the point of weeping at most unexpected times. No physical findings, after a most competent examination at the hands of a number of well-known specialists, could be discovered; and his home and social environment were all that could be desired. He was utterly indifferent to all the usual forms of amusement and displayed a marked aboulia when they were urged upon him. By early and incessant attention to business he had so narrowed his mental horizon that when he began to relinquish business cares and have more leisure time on his hands, he found himself mentally stranded and knew not what to do to entertain himself. His affairs were in a satisfactory state. He traveled extensively. He had no patience with the playing of mere games. He did not like reading and the selections he attempted were most ill-advised. He was thoroughly unhappy without a cause. And yet the cause was amply apparent to a psychologist, though unacknowledged by the patient and his family. It was a case of mental poverty, not insanity.

A brilliant woman, with strong and active literary tastes but inclined to be somewhat erratic, developed a hysteroid state of mind, even to the point of wandering away from her home one night. A very cursory psychoanalysis revealed the fact that her environment, her restraints, and her enforced activities were all in direct opposition to her tastes, modes of thinking and ambitions. A change of environment and a correction of some of her erroneous habits restored her to the condition of a happy and normal existence. She was the victim, not of insanity but most uncongenial surroundings.

A rather amusing case that showed so much psychoneurotic disturbance that her husband brought her to me for examination of her sanity, was that of a woman who understood not a word of her husband's language, he and all of his family being French. The couple were devoted to each other and she always spoke of her husband in the highest terms. Of course he spoke English as well as French. In spite of her "nervousness" and occasional hysteroid outbreaks, I could discover nothing really abnormal with the woman.

After many conferences and a close study of her confidences, I learned that her whole trouble was anxiety, dissatisfaction and injured pride caused by her husband's devotion to his family and her frequent visits with him there, where she sat stupidly listening to conversations of which she understood not a word.

A pampered daughter of wealthy and foolish parents married a successful young business man. The loneliness of her home was so intense to her that she developed a highly psychoneurotic condition. She had periods of mental abstraction when she would wander out of her home. Once or twice in her morning *deshabille* she followed her husband in a daze to his office. All of this, of course, embarrassed him and her family. Many opinions were sought, some of which frankly stated that the case was one of borderland insanity. After the death of the father the girl lived most intimately with her mother, who was a most self-indulgent and undisciplined woman. They slept together, and were never out of each other's sight, day or night. This intimate and foolish association was suddenly broken off when the girl entered her own home as a bride. A prolonged isolation in a hospital from husband as well as family restored the young woman to normal state.

The early history of a young man whom I watched for over a year easily accounted for his marked homosexual tendencies. Vicious education, association and habits entered into the etiology of his acquired homosexuality. Recovery took place in a year under treatment.

Seeing a sister's baby poisoned by rat poison and holding it in her arms when it died so shocked a young woman that long after marriage she refused to have a child of her own. Both she and her husband really wanted a child and were apparently able to have one. Sexual intercourse kept the wife in a constant state of fear. To overcome this the husband refrained. This was again interpreted by the wife as indifference and want of affection on his part. She became highly hysterical and even insanity was spoken of. A judicious psychotherapy and insistence upon a normal sexual life with the hope of begetting a baby have completely restored the woman's health and the happiness of the home. She is now the mother of two vigorous children with not a sign of a psychoneurosis.

A clergyman once came to me on account of extreme bashfulness and distress, when in company, that he was offensive to everybody about him. Being a thinker and a scholar, he wondered whether this indicated some oncoming mental trouble, for he felt sure that there was no real ground for him to feel this way. The history revealed the fact that, like a fish out of water, he was both intellectually and culturally far in advance of the little humdrum, commonplace hamlet in which he was located. He lacked the mental stimulation that comes from intercourse with equals and received, on the other hand, much criticism from those who in their pride resented his almost unavoidable instruction. A long period of this sort of thing in a sensitive individual had at last resulted in the peculiar and distressing self-depreciation for which he came to me. He was a most instructive and entertaining man to meet. Whether my seances with their psychotherapeutic ministrations ultimately overcame the trouble I know not. The patient declared he felt better and I decided that there was nothing of the "borderland" psychosis about the case.

But why go on enumerating more cases? Every practitioner sees them and sometimes to his sorrow. They cause him much loss of time and expenditure of brain matter. Lucky he is if he can always distinguish the real from the apparent psychosis in these borderland cases.

In conclusion, the following propositions seem to me to be worthy of reiteration:

1. Borderland cases are not those that are frankly and definitely recognized to be insane. The usual so-called borderland cases are generally far enough away from the accepted normal to be easily recognized as being really pathological. The adjective borderland should be applied only to those cases which reveal a state of mind out of harmony with the accepted normality of its environment and which may or may not be pathological or, in old terminology, insane, according to the findings of a thoroughgoing physical and psychological examination.

2. The somatic and the psychological manifestations are the only means whereby we can establish the diagnosis of these cases. While the latter are most in evidence usually, the former are the more important, in conjunction with the latter, in establishing the posi-

tiveness of the diagnosis. When the somatic and psychological symptoms are both obtainable the diagnosis of the case is comparatively easy, and resolves itself into a diagnosis practically of the organic or functional disease of which the somatic manifestations are the prime symptoms.

3. There are borderland cases so slight or so insidious that nothing but the mental manifestations appear to be out of the ordinary. The bodily state reveals nothing abnormal, so far as all the modern means of examination can detect. As a rule, these cases are mere *misfits*. They are out of harmony with their entourage. Sometimes they never get into a proper environment and so they always seem queer, a little unbalanced, possibly a borderland psychosis or psychoneurosis. The management of these individuals is obvious.

4. The correct diagnosis of these misfit cases, apparently borderland cases, revealing, as they often do, nothing but psychological eccentricities, is always a matter of relativity. Not only is the patient under critical examination but the examiner and the community are subject to the same relative examination.

5. The diagnosis of a mental state, unlike the diagnosis of any other functional activity of the organism, involves a knowledge of all the workings of the human intellect and emotions, which is a knowledge of the whole of the human race, its history, its productions, and its trends, a vast subject indeed!

6. *Averages* and *normals* are not the same things by any means.

7. We have *average* standards in abundance; *normals* we literally know nothing of. Averages are always relative; normals, in relation to so fluctuating and evanescent a thing as psychosis, are inconceivable. Mind and normality, in the true sense, are incompatible terms. Normal stands for fixity; mind for ever active and reactive variability. An imbecile is normal in a community of imbeciles; a Shakespeare is abnormal in a community of commonplace mentality.

8. Society has a perfect right to legislate what it feels to be a desirable (average) standard mentalization conducive to the preservation of its own welfare; society has no right to declare a mind normal or abnormal, except along the very coarsest, persistent lines, until it includes in its standard the highest, noblest, and most

perfect (which is often unknown to society) degree of mental and moral thinking. What is to determine such a standard, the lowest, the middle average, or the highest types of intellect? It is all a matter of relativity and environment. This is what renders the diagnosis of these real borderland psychoses a task of unusual difficulty.

THE DIAGNOSIS OF ABNORMALITIES OF MYOCARDIAL FUNCTION

By T. STUART HART

Assistant Professor of Clinical Medicine, College of Physicians and Surgeons,
Columbia University; Visiting Physician, Presbyterian Hospital
New York

VI. AURICULAR FLUTTER

Closely allied to "Auricular paroxysmal tachycardia," discussed in the last paper, is an abnormal functional activity of the heart usually designated as *auricular flutter*.* The terms "Auricular tachycardia" (Robinson), "Auricular tachyrrhythmia" (Hoffmann), and "Auricular tachysystole" (Rihl) have also been applied to this condition.

The chief distinguishing feature of this group is the rapid, rhythmic, coördinated systoles of the auricle, the contractions usually occurring at a rate between 250 and 300 per minute. The auricular rate is so rapid that the ventricle is unable to respond to each impulse so that the ventricular rate is always slower than the auricular. The abnormal activity may occur in short paroxysms lasting only a few minutes or may be continued for days or weeks. It seems quite probable that this peculiar activity differs essentially from that of auricular paroxysmal tachycardia only in respect to the rate of the auricular contractions; in paroxysmal auricular tachycardia the auricular rate usually does not exceed 250 per minute and the ventricles respond to each auricular stimulus; in auricular flutter the auricular rate is much faster and the ventricles are unable to respond to each auricular stimulus.

*Jolly and Ritchie, *Heart*, 1910-11, II, 77.

EXPERIMENTAL PRODUCTION

As early as 1887 MacWilliam* described the phenomena which result from the application of a weak faradic current to the exposed auricular wall as follows: "It sets the auricles into a rapid flutter . . . the movements are regular: they seem to consist in a series of contractions originating in the stimulated area and thence spread over the rest of the tissue. The movement does not show any distinct sign of incoördination: it looks like a rapid series of contraction waves passing over the auricular wall." Under these conditions the ventricular rate is accelerated but is usually one-half or less than one-half of the auricular rate. In a heart beating 140 to 180 per minute such faradization may induce an auricular rate of 500 to 600 per minute while the ventricular rate may be 200 to 300 per minute. If faradization of the auricles is stopped the "auricular flutter" may continue for a considerable time and then the auricle may resume its physiological rate.

In the frog's heart "auricular flutter" lasting as long as two minutes, starting suddenly and terminating abruptly, may be induced by a single induction shock applied to the sinus† or some portion of the auricle.‡

While the auricles are in "flutter" vagus stimulation may change the flutter into a condition of "fibrillation" and slow the ventricle; it does not, however, slow the coördinated contractions of the auricle. It is possible, as suggested by Ritchie, that excessive stimulation of the accelerator nerves may be a factor in producing flutter in an otherwise healthy heart.

MECHANISM

Experimental and electrocardiographic evidence indicates that auricular flutter is characterized by a rapid rhythmic series of auricular contractions having their origin in some point of the auricular musculature other than the sinus node. Nearly all paroxysms of auricular flutter are preceded and followed by extrasystoles which interrupt the physiological rhythm more or less frequently; the extrasystoles are auricular in origin and probably arise in the wall

*Journ. of Physiology, 1887, VIII, 296.

†Lovén: Mitteilungen vom physiol. Laboratorium in Stockholm, 1886, IV, 16.

‡Engelmann: Arch. f. d. ges. Physiol., 1897, LXV, 109.

of the upper chamber at a point which becomes the pacemaker for the paroxysm. That the irritability of this point is very great may be concluded from the great rapidity of the auricular systoles. The mechanism is the same as that of auricular paroxysmal tachycardia but in flutter the auricular rate is so great that the capacity of the bundle of His to convey stimuli is exceeded and the ventricle responds only to every second or third auricular impulse. In most cases the ventricular response is perfectly rhythmic and there is one ventricular contraction to two or three auricular contractions. Less commonly the ventricular contractions are arrhythmic and respond at one time to each second auricular impulse, at another time to each third or fourth impulse from the upper chamber.

The activity may be regarded as an auricular tachycardia with a functional depression of the property of conduction in the *A-V* bundle. We conclude that a real depression of conduction exists because we know that in "paroxysmal tachycardia" the ventricle may respond to the auricular impulses at a rate above 230 per minute,

Diagrams to illustrate the mechanism of auricular flutter of different types. The arrows indicate the points of origin and direction taken by the stimuli. Dotted arrows indicate the time at which the normal stimuli at the sinus node should reach maturity if its formation were not interrupted by the abnormal impulse starting from a lower point in the auricle and traveling upward. The thickness of the lines representing ventricular systole indicate the relative effect of the several contractions in maintaining an adequate circulation. The obliquity of the *A-V* line indicates the varying length of the conduction time. *As* = auricular systole. *A-V* = conduction from the auricle to the ventricle. *Vs* = ventricular systole.

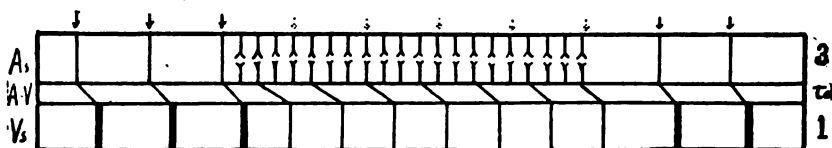


FIG. 1

Paroxysm of auricular flutter. During the attack the auricles contract rhythmically. The ventricles contract rhythmically at a slower rate, the ventricle responds to every third auricular impulse.

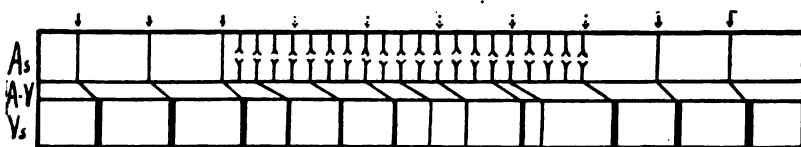


FIG. 2

Paroxysm of auricular flutter with irregular ventricular response. Such an extreme grade of ventricular irregularity may simulate the activity of auricular fibrillation, or a lower grade of irregularity may be mistaken for extra systoles. The ventricle responds to the first, second, third or fourth auricular impulse.

yet in "auricular flutter" the rate of the lower chamber of the heart is usually not above 120; rarely it attains a rate of 160 per minute. Ritchie* has reported a patient with a ventricular rate at times under 40; in this case there was probably an organic lesion of the bundle of His.

Figure 1 shows in diagrammatic form the mechanism of a paroxysm in which the ventricle responds rhythmically to every third auricular impulse; during the attack the ventricular rate is accelerated but is only one-third the rate of the auricle. Each ventricular systole of the paroxysm is less forcible, since the property of contractility has not had the same time to recover as is permitted during the physiological rate. The exhaustion of the capacity of conduction in the *A-V* bundle, due to the abnormal shower of auricular impulses, is indicated by the obliquity of the line representing the period of the passage of the stimulus from the auricle to the ventricle.

In figure 2 are plotted the auricular and ventricular activities of a paroxysm of flutter in which the ventricular response is very irregular; the lower chamber follows the first, second, third or fourth auricular impulse in a seemingly haphazard fashion. The conduction period is variable and prolonged. The ventricular contractions have a force proportional to the preceding diastolic period. The difficulty of differentiating such a mechanism from that of "auricular fibrillation" is apparent. If the ventricular response had been rhythmic up to the time of the final beats of the paroxysm, it is easy to see how the pulse and heart sounds might suggest the occurrence of an extrasystole only.

ETIOLOGY AND PATHOLOGY

The reported cases of auricular flutter indicate that it occurs considerably more often among men than women. It may occur at any age; the earliest subject which has been put on record was 5 years old. All the cases which I have observed, with one exception (14 years), have been over 50 years of age. Ritchie in his analysis of 49 cases found that 70 per cent. occurred after the fortieth year.

*"Auricular Flutter," London, 1914, 36.

Auricular flutter rarely occurs without some other evidence of damage to the cardiac tissues; about a third of the cases show a defect of the mitral valve. Dilatation of the auricles is a common antecedent condition. Pericarditis has been present in several cases. General arteriosclerosis in which the coronaries have participated has been found in a number of instances.

The acute infections, such as diphtheria and rheumatic fever, have been the evident causative agent in about 20 per cent. of the cases thus far reported. Evidence of a syphilitic infection is obtained in at least 10 per cent.

It has been suggested that an abnormal balance of external nervous control may be an element in the production of auricular flutter, but no anatomical lesion which would indicate a removal of vagus influence or a hypertonic activity of the accelerators has thus far been demonstrated.

Such evidence as is at hand leads us to believe that this abnormal activity has its origin in a lesion in the auricular wall which constitutes a focus of increased irritability.

In the few post-mortems which have been reported, in those who have been the subjects of auricular flutter, histological examinations have failed to demonstrate a particular focus in the auricular wall to which one could ascribe the functional change, but general inflammatory and degenerative changes of the myocardium are not wanting. Dilatation of the auricles with fibrous, fatty or lymphocytic infiltration of the walls is the most common finding. Atheroma of the coronaries and calcareous deposits in the arterial wall suggesting an interference with the nutrition of the heart musculature have been found in several instances. These lesions frequently involve a large part of the heart muscle and may include the sinus node and *A-V* bundle. Ritchie (Case III) found changes in the sinus node consisting of lymphocytic infiltration. Hemorrhage and granular degeneration of the nodes are reported by Hume.* I have obtained autopsies in three cases, men of 51, 54 and 55 years, respectively. Each showed sclerosis of the coronaries and extensive fibrous myocarditis; in each very little normal heart muscle could be found. Each had an old infarct of the left ventricular wall.

*Heart, 1913-14, V, 25.

IDENTIFICATION

A careful history and physical examination may lead us to suspect "auricular flutter," but one can only be sure of the correctness of the diagnosis when fortified by the evidence of graphic records. The pulsation of the veins of the neck gives us certain information in regard to the activity of the right auricle, a very rapid rhythmic pulsation of the jugular vein, showing a continuous series of waves at absolutely equal time intervals and two or three times as rapid as the ventricular rate, as determined by auscultation, suggests an auricular flutter, but it is quite evident that by mere inspection it is most difficult to count and correctly determine the spacing of the small venous waves. In cases of established auricular flutter I have repeatedly tried to elicit auscultatory evidence of the rapid auricular activity with complete failure.

The ventricular contractions may be perfectly rhythmic and so accelerated that one may suspect a true "paroxysmal tachycardia" (see paper V). As a rule, in "auricular flutter" the ventricular activity is less rhythmic and not as fast as is the case in "paroxysmal tachycardia." The irregular ventricular activity of "flutter" is most often mistaken for the far more common disturbance known as "auricular fibrillation" (see paper VII). In most cases of "auricular flutter" the arrhythmia is not as great as in "auricular fibrillation" and in the latter the ventricular form of the venous pulse may aid in distinguishing the two conditions; however, without the assistance of graphic records the separation of these groups is practically impossible.

When the ventricular rate is only 40 or less and perfectly rhythmic, one at once suspects a condition of heart block. If in such a case the jugulars are pulsating rhythmically at a rate of 200 or more per minute, one can be reasonably sure that a condition of "auricular flutter" coexists.

There are certain types of irregular ventricular response when the auricle is in flutter which simulate forms of extrasystolic activity. For example, if for considerable periods there is a ventricular response to every third auricular impulse and this established rhythm is broken by a ventricular response to the second auricular impulse, which is in turn followed by a ventricular contraction after four auricular systoles, the early beat and the succeeding pause may

give one the impression of an extrasystole with a compensatory pause (see figure 2).

The *polygram* is often of material aid in making a diagnosis of auricular flutter and the jugular tracing may demonstrate the rapid rhythmic activity of the right auricle. The analysis of the jugular curve is, however, often obscure, since the record of the waves of auricular activity is distorted by the *c* and *v* waves characteristic of the normal venous tracing. We should bear in mind that the *a*, *c*, *v* and *h* waves of the normal jugular pulse do not follow one another at exactly equal intervals of time, and when we can detect in the jugular record such a rhythmic series of waves two, three



FIG. 3

Auricular flutter with regular ventricular response. Auricular rate 276. Ventricular rate 92.

or four times as rapid as the ventricular rate, we have strong grounds for suspecting a condition of auricular tachycardia.

Figure 3 was secured from a case of "auricular flutter" in which there were regularly three auricular contractions to one ventricular. The ventricular rate was 92, the auricular rate 276 per minute. One of the *a* waves of each cycle is simultaneous with the *c* wave. The ventricle contracts in perfect rhythm.

A type of irregular ventricular response is shown in figure 4. The jugular record is composed of a rhythmic series of *a* waves at a rate of 280 per minute, which can be picked out by careful measurement; the pure auricular record is distorted by *c* and *v* waves of each cycle and the whole is superimposed on the respiratory curve.

The ventricle is contracting at a rate of 102 per minute; the ventricle usually responds to the fourth auricular impulse, but occasionally (at X) it responds to the second auricular impulse. This type of irregular ventricular response would strongly suggest occasional auricular extrasystoles were it not for the evidence obtained from the jugular tracing. The analysis of both of these polygrams was verified by electrocardiographic records taken at the same time.

The *electrocardiogram* must be our final court of appeal in substantiating a diagnosis of "auricular flutter." Even here the evidence is sometimes obscure, and it is wise to have records taken by the three standard leads in order to be certain of our interpretation.

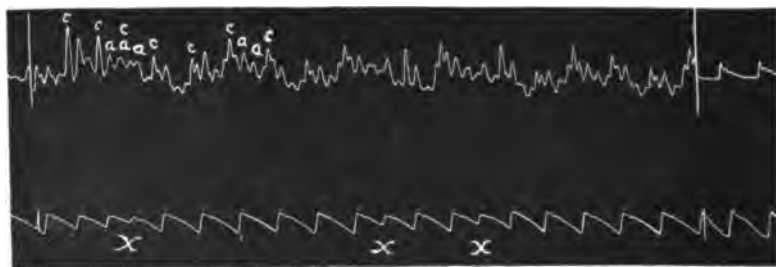


FIG. 4

Auricular flutter with irregular ventricular response. Auricular rate 280. Ventricle usually responds to fourth auricular impulse, at X it responds to second auricular impulse.

Figures 5 (lead I), 6 (lead II), and 7 (lead III) were taken from the same patient at intervals of about one minute and indicate the differences in the records secured by different derivations. Usually the analysis is most easily made from leads II and III, but this is not always the case. In these records the ventricle is beating rhythmically at a rate of 84 per minute; the auricle is contracting at a rate of 336 per minute. One of the auricular (*P*) waves of each cycle is submerged in the *R* deflection of the ventricular cycle. The *T* wave of the ventricular complex is evident only in leads I and II as a slight distortion of the rhythmically recurring *P* waves.

In figures 8, 9, 10, and 11 are shown four records from four distinct cases of auricular flutter indicating the variations which such a group of cases may present.

In figure 8 is reproduced a curve taken from a patient by lead I.

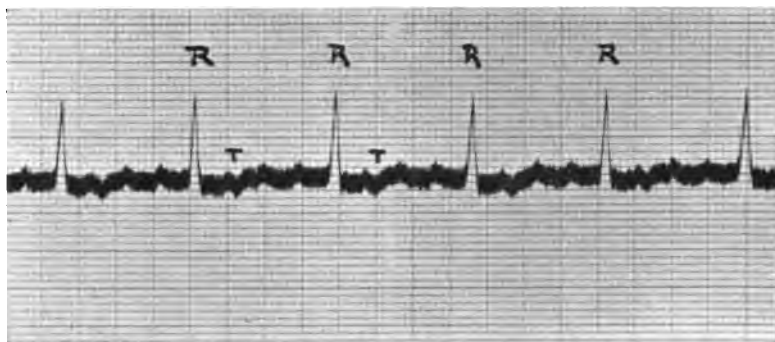


FIG. 5

Patient H. S. Lead I. Auricular flutter. Time lines = $1/25$ second.

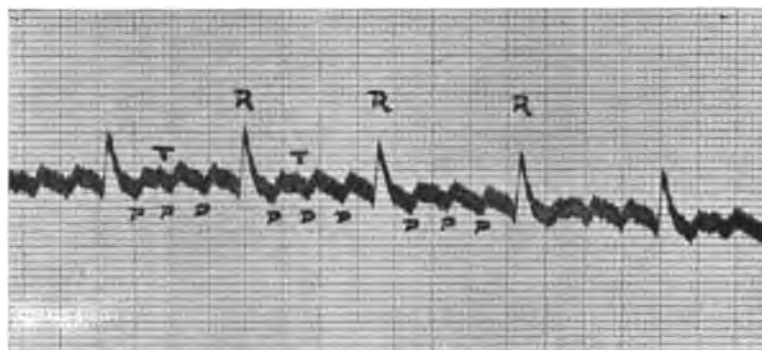


FIG. 6

Patient H. S. Lead II. Auricular flutter. Time lines = $1/25$ second.

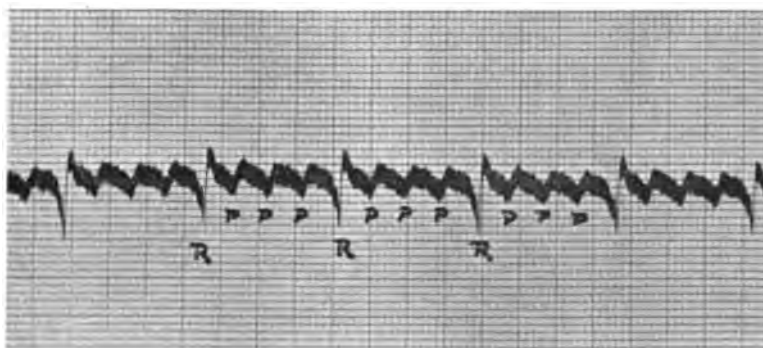


FIG. 7

Patient H. S. Lead III. Auricular flutter. Time lines = $1/25$ second.

Figures 5, 6, and 7 taken from the same subject. Auricular rate 336 per minute. Regular ventricular response to every fourth auricular impulse, ventricular rate 84.

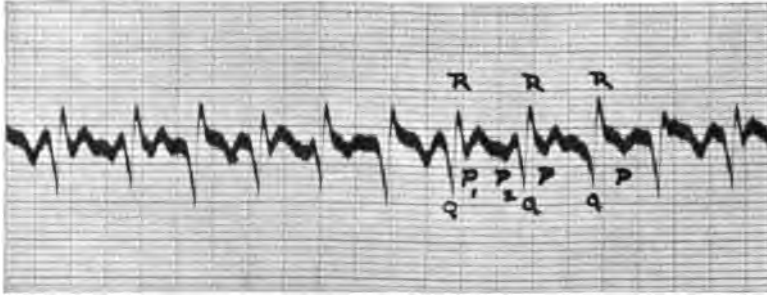


FIG. 8

Auricular flutter. Lead I. Auricular rate 332. Ventricular rate 166, As:Vs::2:1. Time = $1/25$ second.

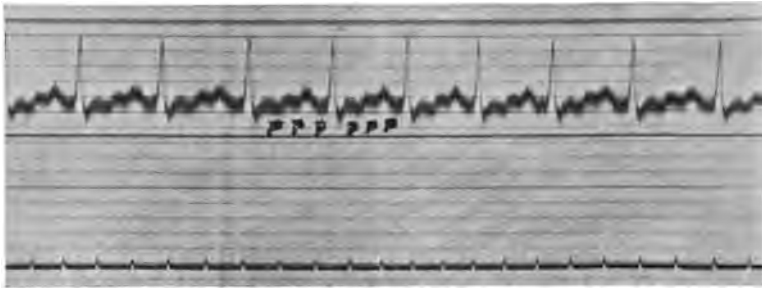


FIG. 9

Auricular flutter. Lead II. As:Vs::4:1. As = 492. Vs = 128. Time = 0.2 second.

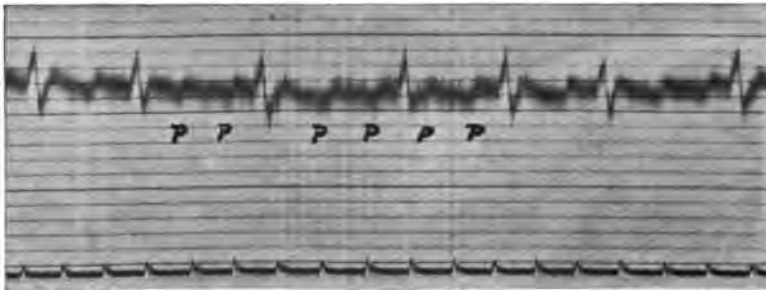


FIG. 10

Auricular flutter with irregular ventricular response. Lead III. As = 280. Time = 0.2 second.

In this instance the ventricular rate is 166; the auricular rate is 332 per minute. The ventricular and auricular complexes are in part superimposed so that the analysis at first glance seems obscure; by the aid of records taken by leads II and III (not reproduced) we could clearly establish a rhythmic rapid activity of the auricle at double the rate of the ventricle. The question arises in this case as to which of the auricular stimuli excites the activity of the lower chamber. We cannot answer this question positively but we have strong evidence for presuming that the *earlier* of the auricular stimuli (P_1) is the one to which the succeeding ventricular contrac-

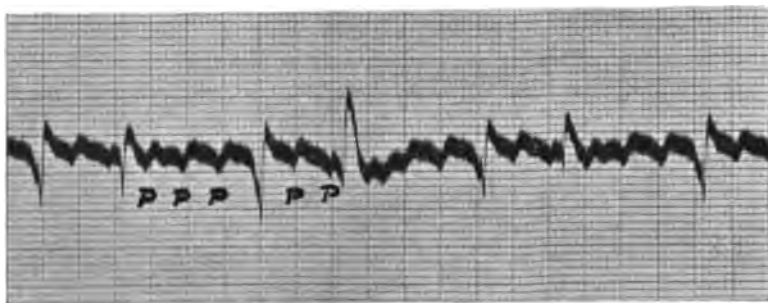


FIG. 11

Auricular flutter. Lead III. Irregular ventricular response. One ventricular extra systole. Time = $1/25$ second.

tion is the response. If the response was to the stimulus delivered at P_2 the conduction time (P_2-Q) would be abnormally short. While it is not inconceivable that in certain cases the property of conduction may be heightened, all our experience goes to show that in those cases of auricular flutter in which we have positive evidence, conduction is normal or depressed (usually the latter). It is never demonstrably shortened, hence we are led to believe that in every case the conduction time is longer than the normal and therefore in the instance shown in figure 8 it is probable that the ventricle responds to P_1 rather than to P_2 .

A case in which the lower chamber response follows four auricular contractions is depicted in figure 9. The ventricular rate is 128; the auricular rate is 492. Both chambers contract rhythmically but the auricle four times as often as the ventricle.

Irregular ventricular responses are shown in figures 10 and 11. In figure 10 the auricle is beating rhythmically 280 times per minute; the ventricle responds to every second or third auricular stimulus.

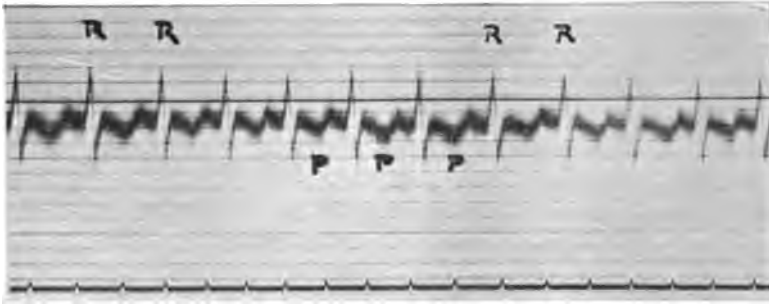


FIG. 12

Auricular flutter. Patient H. Auricular rate 388. Regular ventricular response rate 194. Time = 0.2 second.

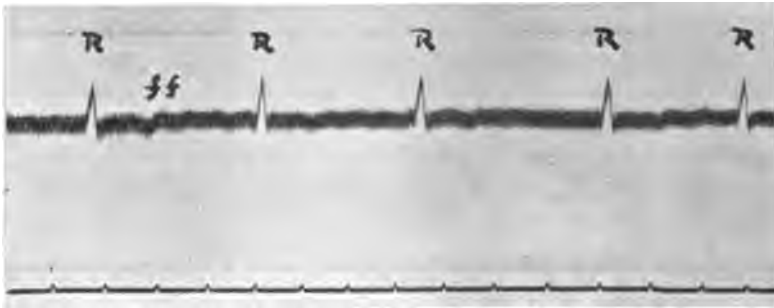


FIG. 13

Auricular fibrillation. Taken from same patient (H.) as figure 12, but 15 days later. Time = 0.2 second.

In figure 11 the response is to the second, third, or fourth auricular stimulus. This record is further complicated by an unusual complex indicating one ventricular beat having its origin in a point in the ventricular wall quite different from the other ventricular contractions, which are of supraventricular origin.

A record from a case of "flutter" with a very rapid rhythmic response is represented in figure 12. The auricular rate is 388; every

other auricular complex is submerged in a ventricular complex which occurs 194 times per minute. The ratio of the rate of the upper to the lower chambers is as 2 is to 1. A record from the same case (figure 13) taken 15 days later, after the patient had taken digitalis, shows complete irregularity and a rate of 46 per minute. There are at this time no coördinated contractions of the auricle, but its activity is one of "fibrillation."

CLINICAL COURSE AND SIGNIFICANCE

Auricular flutter is occasionally the only evidence obtainable of a defective myocardium, though quite commonly extrasystoles precede and follow the paroxysms. In such patients careful examination fails to reveal any organic change in the valves, endocardium or pericardium, and the only evidences of functional disturbance are those elicited during the paroxysm. During the attack, which comes on abruptly and terminates suddenly, the patient may be very uncomfortable. He is conscious of an unusual commotion in the chest; the accelerated and irregular activity of the ventricle may be the cause of considerable apprehension; this may be accompanied by some dyspnoea, precordial distress and prostration if the paroxysm is prolonged. Some attacks may extend over days or even weeks, the earlier alarm and dyspnoea may subside, and the patient may resume his usual occupation aware only of the continuing "palpitation."

In most cases there are other evidences of myocardial damage and the "auricular flutter" throws an additional load on a heart already overtaxed. In such patients the general signs of cardiac insufficiency may have been present before the onset of the auricular acceleration, or the unusual stress occasioned by the new rhythm may be too much for a heart barely able to preserve an adequate blood stream; its narrow margin of safety is quickly exhausted, and signs and symptoms of cardiac insufficiency rapidly appear. The extent and severity of the symptoms depend to a very large degree on the condition of the heart before the attack; the auricular flutter may last for days or weeks, yet ultimately the heart may recover a normal rhythm and perform its work with reasonable efficiency; or in a short time there may develop dyspnoea, congestion of the liver

and lungs, edema of the extremities, Cheyne-Stokes respiration, giddiness, unconsciousness and collapse.

A patient may have many attacks of auricular flutter or it may appear only as a terminal event. Once established, the attacks are

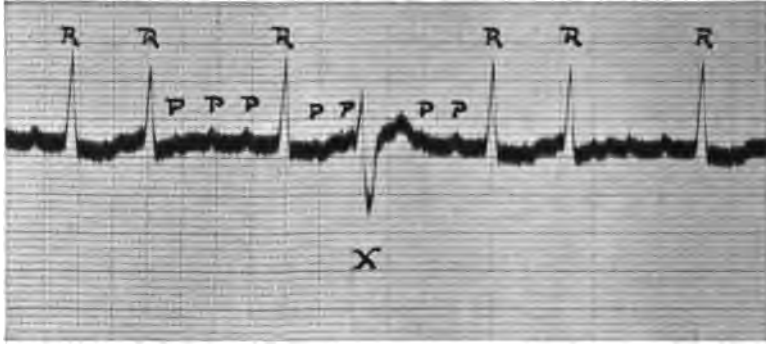


FIG. 14

Patient K. Auricular flutter with irregular ventricular response and ventricular extra systole. Time $1/25$ second.

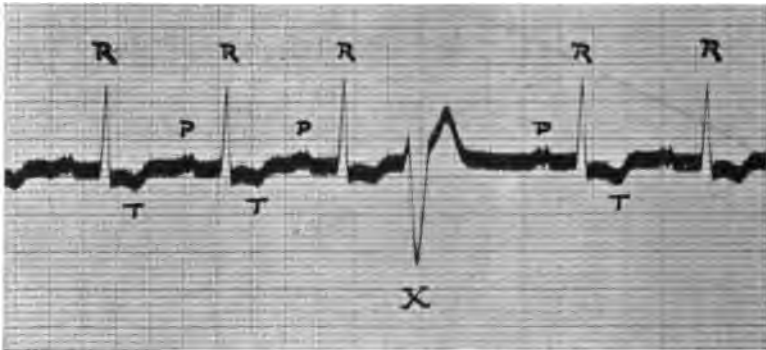


FIG. 15

Patient K. Sequential rhythm taken from same subject as figure 14. Taken 11 days later. Note same type of ventricular extra systole. Time $1/25$ second.

prone to recur and each one is apt to persist for a longer time. Occasionally one sees attacks of flutter alternating with periods of normal rhythm; more often "auricular flutter" passes into "auricular fibrillation," which may persist indefinitely or may, in turn, give

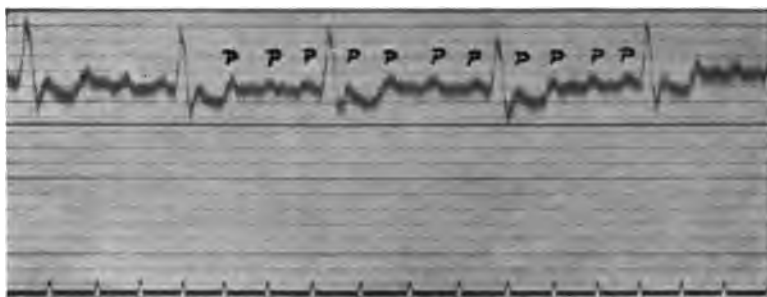


FIG. 16

Patient M. K. March 11, 1912. Auricular flutter. Time 0.2 second.

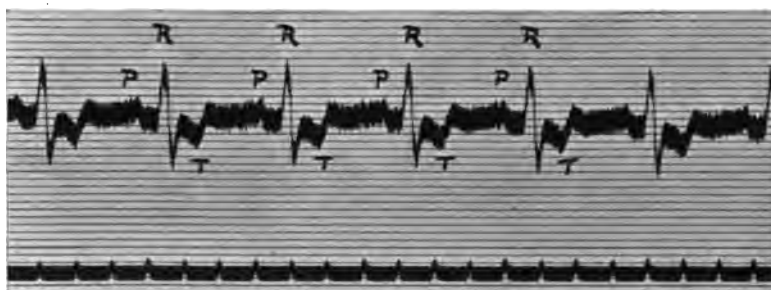


FIG. 17

Patient M. K. December 12, 1914. Sequential rhythm. Time 0.2 second.

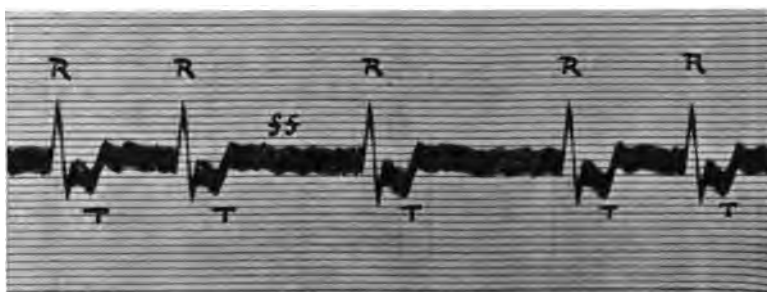


FIG. 18

Patient M. K. December 28, 1914. Auricular fibrillation. Figures 16, 17 and 18 are taken from the same subject, the first of these was taken during an attack of pneumonia 2 1/2 years before the subsequent records.

way to a physiological rhythm. With a return to a normal rhythm the symptoms usually improve.

The tendency to resume a normal rhythm is seen in figures 14 and 15, taken from the same patient at intervals of eleven days. Figure 14 shows auricular flutter at 300 per minute with an irregular ventricular response interrupted at X by a ventricular extrasystole. In figure 15 is seen the sequential rhythm of eleven days later interrupted by an extrasystole of the same type as that which occurred during the period of "flutter."

Figure 16 was taken from a patient during her first paroxysm of flutter, which had its onset during an attack of lobar pneumonia in March, 1912. In December, 1914, she returned to the hospital with broken cardiac compensation. Her record taken at that time (figure 17) shows a sequential rhythm. A few days later she began to fibrillate (figure 18) and has continued this condition up to the present time (6 months later).

The clinical significance of auricular flutter lies in the fact that it indicates a considerable degree of damage present in the auricular wall. That the damage may be temporarily repaired is indicated by the recovery of normal rhythm, but the tendency to repeated and more severe attacks suggests that usually the repair is incomplete.

The welfare of the patient depends to a large degree on the condition of the ventricle. With a normal ventricular muscle the patient will withstand many attacks of "auricular tachycardia" with comparative immunity. With a damaged ventricle the outlook is much less propitious. Unfortunately the myocardial damage is rarely limited to the auricle. In "auricular flutter" a slow, regular response of the ventricle is favorable; a rapid, irregular ventricular response makes the outlook more serious. The change to a condition of auricular fibrillation and a slowing of the ventricle under digitalis are to be regarded as a favorable sequence of events. The return to a normal rhythm is to be welcomed but by no means assures complete recovery.

HYPOADRENIA MISCALLED NEURASTHENIA.
DIAGNOSIS AND PROGNOSIS

By TOM A. WILLIAMS

Neurologist to Epiphany Dispensary and Freedmen's Hospital; Lecturer on
Nervous and Mental Diseases, Howard University; Corresp.

M. Soc. de Neurologie de Paris, etc.

Washington, D. C.

Twenty years ago the name neurasthenia was satisfactory to general practitioners and to neurologists, the disease being characterized by weakness of muscle, nerve and mind.

A disease in which this weakness was extreme, and usually fatal in from a few weeks to ten years, was described by Addison in 1855, and was found to be due to partial destruction of the suprarenal glands.

But besides Addison's disease there are many degrees of insufficiency of these glands; and we are now beginning to comprehend that many symptoms long observed heretofore are due to lesions thereof, causing reduction in the quantity of adrenal secretion and hence hypoadrenia.

As the writer has said previously,* the term neurasthenia amounts to nothing more than a convenient cloak for failure to investigate a case sufficiently, and in speaking of hypoadrenia as a cause of neurasthenia it is desired to call attention to the parallel existing between what has been called neurasthenia and the present conception of hypoadrenia.

Various authors have given the following as some of the symptoms of neurasthenia: failing strength, prostration after exertion, cold extremities, anemia, constipation, loss of control of attention, weakness of memory, weak will power, inability to perform mental work, incapacity of decision, abdominal throbbing from pulsating aorta.

Compare the now well-known symptoms of hypoadrenia: asthenia, sensitiveness to cold and cold extremities, hypotension, weak cardiac action and pulse, anorexia, anemia, slow metabolism, constipation, sometimes psychasthenia, as well as others which appeared in the cases reported herewith.

*Archives of Diagnosis, 1909. Differential Diagnosis of Neurasthenia.

Hypoadrenia may result from the wasting of old age, the toxins of the infectious diseases, hemorrhages into the substance of the gland due to high blood-pressure, or perhaps from exhaustion by long-standing emotions.

So that neurasthenia, we may realize, is hypoadrenia.

We know something of its pathology, but no one ever knew of a satisfactory pathology of neurasthenia. The symptoms of hypoadrenia stand out clearly, which never could be said of neurasthenia; and armed with this more definite knowledge, we can formulate and carry out better ideas for its treatment.

An instructive case is that of a mechanic fifty-seven years old, reported elsewhere,* whose history showed malaria as a cause of his adrenal deficiency, and whose subsequent symptoms might easily have lead to a diagnosis of neurasthenia. He felt unable to work because he was weak and dizzy, he had that throbbing in the abdomen which has been mentioned as a symptom of neurasthenia, and asthenia was present in marked degree. But he also had a slight tremor of hands and face, a hint of von Graefe's sign, and hypotension, his systolic pressure being 108. It was clear that hypoadrenia was the cause of his trouble, and adrenal substance cured him in two weeks.

A still more interesting example was an official in a responsible position whose mental depression, asthenia, languor, flabby muscles, and incapacity of decision made a very good picture of neurasthenia, and he feared dementia. There was some tremor. Hypotension was evident, systolic pressure being 100 at forty years of age.

Adrenal therapy improved him so that he was able to accomplish the work that he formerly could not do. Later he reduced the dose until able to go without it.

A woman who had a postpuerperal psychosis, attacks of melancholy, had lost interest in life, food had no taste, was in despair because she wished to cease child-bearing. This might have been called neurasthenia, but there was brown pigmentation of the skin and a systolic pressure of 112, which soon fell to 90. At first 4 grains daily of adrenal substance was given without any visible effect. The dose being doubled, however, improvement was rapid,

*Jour. A. M. A., November 9, 1913.

and afterward the dose was successfully reduced. This result confirmed the diagnosis of hypoadrenia.

A professor of economics had headaches for six years, worse after using eyes, had soreness over whole body, especially the neck, dull pain over sacrum, and a tired voice. Oculists had failed to relieve him. He felt disinclined to take exercise, a change from his former habits, and he had no sexual desire. A diagnosis of neurasthenia would have seemed appropriate. He had scarlet fever in his youth, and an attack of the grippe had greatly aggravated his symptoms, and these toxic sources of adrenal weakness suggested adrenal therapy, though hypotension was very moderate, systolic pressure being 114. He improved on small doses, and was afterwards able to do effective work if it was limited to four hours a day. He has later recovered completely.

A man of forty who was soon to be married feared he was impotent, and worrying about this had run him down until he was in despair. He was depressed and was quickly fatigued, and was found to have a pigmented abdomen and a systolic pressure of 102. He was given adrenal substance and fully recovered, as a result of this and appropriate psychotherapy to prevent the emotional stress to which he had subjected himself.

These cases were all referred to me by fellow practitioners as neurasthenics previous to about three years ago. Since then, a number of others have been brought me, but to relate them would merely be a repetition of the facts.

Some cases do not do as well as these which have been mentioned, however, and an instance is seen in an ethnologist thirty years of age, who felt that his mind was shadowy and nothing seemed real.

He had intestinal flatulence, especially after worrying, sometimes foul tongue, indigestion, dizziness and morning headaches from autointoxication, constipation and sudden insomnia. A temporary gain was soon lost after taking cold. His systolic pressure was only 102, so he was given adrenal substance, in small doses at first. There being no rise of blood-pressure, the dose was gradually increased until he was taking 14 grains a day, with varying pressure, and still feeling discomfort. His improvement was slight, but he worried less and the treatment was continued, in the hope that he would learn to tax himself less in his work, and that thus his func-

tional adequacy would increase. The patient fully recovered after a year.

The fact that some instances of hypoadrenia are commencing Addison's disease, and are destined to terminate fatally when the adrenal glands are destroyed, makes it conspicuously necessary to have a clear knowledge of the real conditions that cause the symptoms, as a diagnosis of neurasthenia under such circumstances would be unfortunate.

Thus the daughter of a Boston physician began to tire easily at study and at play. A long rest was prescribed and she took a long trip to Europe, but the asthenia increased and finally became extreme, the bronzing of the skin decided and the pupils dilated. A diagnosis of extreme hypoadrenalism was made, adrenal substance was given by the mouth and under the skin, but the patient died a week after the treatment was commenced. At autopsy the cortex of the adrenal glands was found to be nearly all destroyed, only part of the medulla and the capsule being left.

Another case was that of a married woman of thirty-four who had a nervous breakdown from overwork. She lost weight, had anemia and asthenia and also feeble heart. Dilatation of the stomach, sunken eyes, pale mucous membrane and râles in the chest suggested tuberculosis. There was brown pigmentation of the skin and a systolic pressure of 86. She died a few weeks later, and as necropsy was refused it is impossible to say whether the failure of adrenal secretion which caused her death was due to tuberculosis or was a simple atrophy as in the preceding case.

My object has been to show by these instances that when we are confronted with patients who have great asthenia, lack of concentration, and more or less of the long list of symptoms heretofore assigned to neurasthenia, we must not be content to stop with the latter as a diagnosis. If, in addition, there are low blood-pressure, subnormal temperature, pigmentary changes, hypoadrenia may be the cause of the depression.

I feel sure that this syndrome is much more frequent than has been suspected. In a special neurological practice by no means large I have already seen upwards of a score of such cases.

In spite of the rapid recovery of some cases, it is not wise to diagnose hypoadrenia therapeutically, for some patients do not improve

for long periods. To give adrenal substances for long is unsafe, as over-stimulation of vegetative functions and internal secretions might ensue. Even if no direct ill effects occurred, however, valuable time would be lost were the patient's neurasthenic state due to another condition than hypoadrenia.

The diagnostic criteria were stated as follows in my paper to the American Medical Association in 1913:

Asthenia.—From conditions of physical causation characterized by great fatigability and feeble circulation the diagnosis would of course be made by the current procedures of clinical medicine, on which I need not enlarge here.

Psychasthenia.—Patients of the type I have described are often labeled psychasthenics because of their feeling of inadequacy, the most prominent feature in psychasthenia. But the genesis of this feeling in hypoadrenalism is a real physical tire occurring only during exertion, while the genesis of the inadequate feeling in psychasthenia is from a besetment or apprehension, and it always passes off while the patient is actively exerting himself with muscles or mind. If there is any feeling of panic because of the dread of his incapacity in a hypoadrenal patient, this is a purely secondary and normal reaction to physical fact; and it is very easily dealt with by enlightenment, as my cases illustrate. Furthermore, psychasthenics present a vast series of intellectual, emotional and often motorial symptoms, the combination of which is characteristic, and the source of which can be detected by analysis of the psyche.

Hysterical Nosophobia and Its Consequences.—The seeming exhaustion and asthenia so often found as a result of a patient's belief in the malfunctioning of a stomach, heart, intestine, limb or brain is a phenomenon of mental attitude induced in the same way as is the case in the little child who suddenly becomes tired when his work or play is no longer to his liking. It is merely a lack of the toughening of "psychologic fiber" necessary to social welfare. It is a phenomenon induced by suggestion, and its name is hysteria. The real physical exhaustion which may occur after long continuance of hysteric attitudes like this is secondary to the depressing emotions sometimes induced in the patient's mind.

From melancholia the incapacity of hypoadrenalism is distinguished by the absence of the retardation of thought and movement

always present in the true melancholic, in whom, furthermore, the responsiveness increases in speed and effectiveness as the day goes on and when the stimuli are augmented and longer continued, whereas in hypoadrenalism the patient is at his best early in the day and the more work he does the worse he becomes.

Concerning neurasthenia, it should be evident that I have ceased to recognize any such nosologic rubric, regarding the term merely as a convenient cloak for failure to investigate a case sufficiently, and placing all the patients who were thus formerly diagnosed in categories corresponding to the etiologic factor which has determined their condition.

A NEW SYMPTOM OF ULCER AT OR ABOUT THE DUODENAL PORTION OF THE PYLORUS

By HEINRICH STERN

New York

The symptom described hereafter is, as far as I could ascertain, not alluded to in literature. Yet it occurs very frequently, as it was present in practically every case of ulcer at or about the duodenal portion of the pylorus that has come under my observation during the past year. The symptom is, without doubt, characteristic of duodenal ulcer, and all but one of the patients exhibiting it were men. In addition to this particular symptom the same patients complained of other symptoms pointing to an ulcerative process within the duodenum, as, for instance, the hunger-pain, and they usually showed most of the signs as disclosed by a chemical and physical (including the röntgenological) examination.

The symptom is both of an objective and subjective nature. It is subjective because it is a reaction to pyloro-epigastric discomfort or pain; it is objective because it is a visible evidence that the patient voluntarily or involuntarily assumes that posture which gives him most comfort.

This posture constitutes the sign or symptom. The patient, trying to stretch out his epigastrium on account of the relief obtained thereby, often prefers standing to sitting; when in discomfort while resting upon a chair he sits in a slanting position in such a manner that chest, abdomen and legs form a perfect incline. When ex-

periencing discomfort while in bed, the patient tries to lie as straight as possible, often on the left, but never on the right side.

The patient, of course, will always speak of his discomfort or pain, but will hardly ever mention the posture he assumes when in distress unless he be directly questioned about it. This is readily understood when we consider that many patients stretch the pyloro-epigastric region in an entirely instinctive manner. (When in the physician's office, the patient naturally feels under a certain restraint, and he does not assume the posture which gives him most comfort.)

It seems almost as if the patient with a duodenal ulcer occupies by preference the posture which is the reverse of that by which he has acquired the lesion. With very few exceptions, the body of the individual during the process of development of a duodenal ulcer, especially when at work, is bent over, the curve being in the pyloro-epigastric region. Very often this region impinges upon a hard object, as a working table, for instance. This is especially the case in right-handed people. (I have never seen a case of duodenal ulcer in left-handed persons.) This curve is bound to interfere more or less with the blood supply of the peculiarly-shaped duodenum, and the production of a localized necrotic spot, commonly styled an ulcer, is not difficult to explain. (Corsets worn by women are apt to prevent this injury.)

On this occasion I shall not attempt to discuss why the drawing out of the body, its stretching, is pathognomonic of ulcer about the distal and not about the proximate side of the pylorus. It probably finds its explanation in the anatomic conditions and relations of the duodenum.

The symptom or sign is of especial value in differential diagnosis. It is not present in gastric ulcer. In gall-stone colic, the patient usually lies on his left side with legs drawn up. In appendiceal colic, as well as in the common affections of the cecum and the colon in general, the body of the patient, when sitting, is bent over; when lying, his legs are more or less flexed.

RARE FORMS OF PERICARDITIS

By GUSTAV SINGER

Vienna, Austria

Despite a number of refined physical methods, among which radiology is a very welcome ally, the clinical recognition of the inflammatory processes of the pericardium leaves much to be desired. It may even happen that these processes are only recognized at the autopsy table. This is hardly astonishing in dry pericarditic affections with fleeting intravital symptoms; however, cases of pericardial exudate may be clinically overlooked by the most experienced and painstaking examiner.

The importance of the inflammatory diseases of the pericardium justifies the thorough revision of all the criteria of an exact diagnosis. In this communication, however, I wish to discuss some forms of pericarditis which are little known, but are of great import in diagnostic as well as prognostic respects.

The relationship of inflammatory processes of the pericardium to stenocardia gives rise to a unique clinical syndrome. It is quite plausible that certain clinical points of connection appear in this symptom-complex. We know, for instance, that acute pericarditis without premonitory symptoms is sometimes associated with a dull, occasionally quite violent pain about the anterior chest-wall. In case a noticeable appearance of apprehension ensues in addition to the precordial pain (a frequent factor in acute cardiac disease) a symptom-complex resembling that of angina pectoris may be produced.

I shall particularly discuss those cases in which, in previously entirely healthy persons, the typical picture of the stenocardic attack is produced suddenly like in angina pectoris. In these cases the attacks may repeat themselves or may continue for days until the appearance of friction sounds in the characteristic location or until the production of the pericardial exudate, phenomena which leave no doubt that the patient suffers from a pericarditic affection.

PERICARDITIS ANGINOSA

This form of pericarditis, as some believe, may be ushered in with initial symptoms which entirely resemble angina pectoris. In

other words, angina pectoris may not necessarily point to coronary disease, but it may fully develop in the form of the symptom-complex of pericarditis. Another group of authors is of the opinion that stenocardia and coronary disease are an inseparable pathological unity, and declares that pericarditis (even the acute febrile form of the same) appearing together with stenocardic attacks, is the result of certain affections of the coronary arteries, especially of thrombosis with following myomalacia. According to this assumption, the pericarditis arises as a secondary inflammation, on account of the involvement of the visceral portion, the epicardium.

It is especially Kernig who has reported a series of cases of pericarditis following stenocardia, and who has endeavored to show that an occlusion of the coronary arteries was at the foundation of each particular instance.

Despite the convincing and anatomically verified conditions, the temporary combination of stenocardia and inflammatory pericarditis is not always caused by coronary disease. There are cases with sound coronary arteries in which the syndrome "pericarditis-angina pectoris" is a characteristic manifestation of the inflammatory cardiac pain. The following case may serve to illustrate my contention.

A man, forty-five years old. Was examined by me previously and found clinically entirely well. The day before he visited me he had, on going into the cold air from a warm place, an attack of very violent cramp-like pains in the chest, together with much oppression and dyspnea. He could not proceed and was brought home by a physician, who described the attack as a typically stenocardic one. The examination showed some pallor, no abnormality of the pulse or the size of the heart. A systolic and a diastolic murmur were noticeable at the apex; at the base, however, the sounds were clear and not accentuated. The liver was somewhat enlarged. Tonometric measurements showed 125 mm. in the morning and 115 mm. in the evening. The Röntgen examination showed a dilatation of the shadow of the aorta at its root and an enlargement of the left auricle. The thorax during inspiration showed superficial excursions, and the patient complained of vague pains corresponding to the location of the diaphragm. During the next few days no temperature elevation was observed. The blood pressure, however,

was increased; the pains continued, and a phenomenon which was present right from the beginning of the attack, *pulsus paradoxus*, was observed.

The intensity of the symptoms of the first attack was never repeated. Continued observation of the patient showed friction sounds in the left mammary and axillary lines. Synchronously with these there ensued violent pains in the thoracic musculature, of the muscles in the extremities and in the articulations of the shoulders and hands. Fever was never present. The muscular pains occurred again during the next two years, and it was finally admitted by the patient that he had always had similar pains in the spring and autumn. The liver became smaller, the *pulsus paradoxus* and the abnormal sounds disappeared. An affection of the aorta or the peripheral blood vessels could never be determined. The patient, an alpine climber, never gave up this sport, and at no time could any affection of any part of his circulatory apparatus be detected.

I do not doubt, though a strict proof has not been furnished, that in this case there had existed a rheumatic, latent pericarditis and mediastinopericarditis (*pulsus paradoxus*) for some time until the sudden acute attack resembling angina pectoris ushered in a frank state of the condition, and that for this reason the stenocardia was not associated with a vascular affection, but was merely a symptom of dry pericarditis. The enlargement of the liver, which disappeared after a few weeks without medication, is of some import as a symptom of pericarditis.

A case of Andral, which is cited by a number of authors, deserves brief mention in this place. The patient was a man, thirty-one years old, who was affected with acute articular rheumatism. On the second day of his disease he had violent pains lasting the entire night and the following day. At the same time there existed arrhythmia, tachycardia with intermissions, small pulse, most pronounced anxiety, pallor, and cold extremities. After twenty-nine hours of this agony the patient died. Necropsy showed that the entire internal surface of the pericardium was covered with a white, membranous exudate beneath which the pericardium was strongly reddened. The organ was filled with an ounce of greenish-serous fluid. The heart, the endocardium, the large vessels, the abdominal organs and the brain showed no pathological alterations.

The cause of the stenocardic pains in pericarditis is, according to Pawinski, due to an affection of the cardiac plexus which is composed of the fibers of the vagus and the sympatheticus. It was Neusser, however, who put into the proper light the appearance of pericarditis under the symptom-complex of angina pectoris. He says that acute exudates into the pericardium may like hematomata call forth a dull precordial pain. This confirms the observations of French clinicians concerning the occurrence of neuritis of the phrenic nerve in such cases.

As far back as 1891, Curschmann has shown that in initial cases of arteriosclerosis calcium deposits at the roots of the coronary vessels may engender ischemia and stenocardic attacks. These findings served Pawinski as well as Neusser to explain those stenocardias in which the lumen of the coronary arteries is compressed and stenosed. However, there are rare cases of angina pectoris causing sudden death in which necropsy only demonstrates a synechia of the pericardium without a narrowing of the lumen of the coronary vessels.

The stenocardias with intact coronary arteries, of which quite a large number are reported in medical literature, form the foundation of the neuritic theory of angina pectoris. In this connection it is of great importance what Neusser has stated in regard of the production of angina pectoris. He says: "Angina pectoris can not only be explained because of ischemia of the cardiac muscle, and its production is not solely caused by the mechanical narrowing of the vessels. Besides the stabile factor of the anatomical stenosis of the vascular lumen there exists a labile factor, *viz.*, an active vasoconstriction due to hypertrophy of the muscularis. Besides, there may be an increased irritability of the nerves and ganglia situated in the vascular walls."

The many authoritative investigations have definitely demonstrated that pericarditis sicca or exudativa may be associated with unmistakable stenocardia. Especially those cases of stenocardia are suspicious of latent or not demonstrable pericarditis, in which the anginal attack is not paroxysmal and brief, but enduring and appearing as status anginosus; cases in which the attacks ensue in early life when arteriosclerosis has as yet not supervened, or which are associated with or intercur during rheumatic affections; cases

in which syphilis may be excluded and in which the violent pain during the first few days is soon coupled with elevation of temperature.

The proper interpretation is often dependent upon the audibility in the typical locations of the friction sounds which are often quite transitory. If these sounds are not heard, many instances of the affection will never be recognized. Generally speaking, frequent, regularly appearing, stenocardic pains supervening for a rather protracted period in the absence of gross changes (dilatation of the aorta, aneurism, demonstrable aortic disease, pronounced arteriosclerosis) point to latent pericarditis.

Patients who are supposed to suffer from angina pectoris often state that they feel quite well when at rest and feel no oppression whatever, but complain that the slightest effort at walking, especially in the street (even in mild weather) compels them to stop. They experience no difficulty in breathing, but assert that an undefinable sensation constrains them. This symptom, in my opinion, strongly points to the presence of pericarditis.

A woman patient, whom I am treating for ten years for an old mitral stenosis and insufficiency, showed a forementioned phenomena. During a night, while in the country for the summer, she had sudden attacks of violent constriction pains in the chest which were pronounced to be of an anginal character by the local physician. The symptoms were repeated with greater or lesser intensity. A disease condition developed for some weeks which was principally characterized by depression, feeling of anxiety and vague phenomena pointing to the heart. During this time I saw the patient once, but could not determine any change from her physical condition previous to the attack. Pronounced symptoms of decompensation were not present. After some weeks I was again summoned, but the patient's condition had not undergone any material change. However, no progress in the subjective condition was noticeable. She complained that, without feeling very sick, she was compelled to lie in bed as she would break down after having made a few steps on account of a feeling of weakness and severe oppression in the chest. For the first time I found a distinct change in the physical findings. There were loud scraping friction sounds over the entire heart, and the attending physician corroborated that the patient had had peri-

carditic friction for some time, but that the resemblance of the first attack to stenocardia had been very great. Strict rest in bed was ordered, after which subfebrile temperatures ensued. Antiphlogistic treatment, etc., caused relief after a comparatively short time. The patient has never had a stenocardia-like attack since.

UREMIC PERICARDITIS

In fully developed uremic conditions circumscribed friction sounds, localized about the base of the heart, are nearly always audible. These sounds are such a regular phenomenon that I look upon the occurrence of dry afebrile pericarditis in vague cases as a pathognomonic symptom of uremia.

Associated with this symptom there exist mostly the characteristic uremic disturbances of respiration and the peculiar symptom first described by Drasche in cholera typhoid, *viz.*, a mealy deposit upon the skin, especially upon forehead and the nose, consisting of very fine urea crystals. The appearance of this symptom seals the gloomy fate of the patient. In numerous cases that perished I have examined the fibrin deposit of the pericardium, which is invariably sterile, for the presence of urea, and I was always able to demonstrate a large number of crystals by the addition of nitric acid. In these cases we have to deal, therefore, with a retention toxicosis of the skin and the serous membranes.

In this form of dry pericarditis the first appearance of the friction sounds over the base of the heart is also characteristic. In case the fibrous exudate in the region of its primary location is but small or the conditions for the production of friction sounds are not sufficiently favorable, the physical, often evanescent, signs of dry pericarditis may not be present at all. Still in such cases the consequences of the compression of the vascular roots by the exudate, especially about the roots of the coronary arteries, may supervene in a form completely agreeing with the typical signs of stenocardia.

The conditions favoring this phenomenon undoubtedly exist quite frequently. This anatomical localization is, as has been shown, a possible explanation of the association of pericarditis and angina pectoris.

DIAGNOSIS OF GENITOURINARY CONDITIONS IN
WOMEN BY MEANS OF THE RÖNTGEN RAY.

BY G. S. PETERKIN

Seattle, Washington

Every one of us knows that the minds of the highest and the lowest of mankind are compounded of the same elements, held subject to the same laws of action; that the knowledge any one of us possesses must come through the ordinary channels of sense—sight, hearing, smell, taste, touch, and the muscular sense.

The correctness of the perception of an object obtained by an individual through his senses is usually in proportion not alone to the accuracy of any one sense, but to the number of senses used to corroborate the primary impression made upon his brain. To prove the truth of a perception to others, means of making an exact objective record must be devised. Then the perception will be accepted and can be used as a means of comparison, making knowledge scientific, instead of speculative. This psychological axiom is clearly expressed in the slang phrase, "Show me!"

It is the purpose of this article to show that a method of making the sense of touch—palpation—a matter of record has been developed, and the diagnosis of genital and urinary diseases in women has been placed, therefore, on a scientific basis, instead of being almost wholly speculative.

The means to this end is an X-ray pessary. The technic of its employment will be briefly presented, with evidence to substantiate the statements made as to its utility.

How this instrument can accomplish its object will be better understood when we call to mind the following facts:

1st. That the ureters, as they pass along the cervix between the broad ligaments, are virtually firmly attached to this organ (Fig. 1, a).

2nd. That practically the entire base of the bladder is firmly attached to the body and cervix of the uterus for at least one and one-half inches (3.7 cm.) (Fig. 1, b).

3rd. That the bladder is attached to the vaginal wall, the anterior-superior portion of which, in turn, receives its support directly from the uterus (Fig. 1, c).

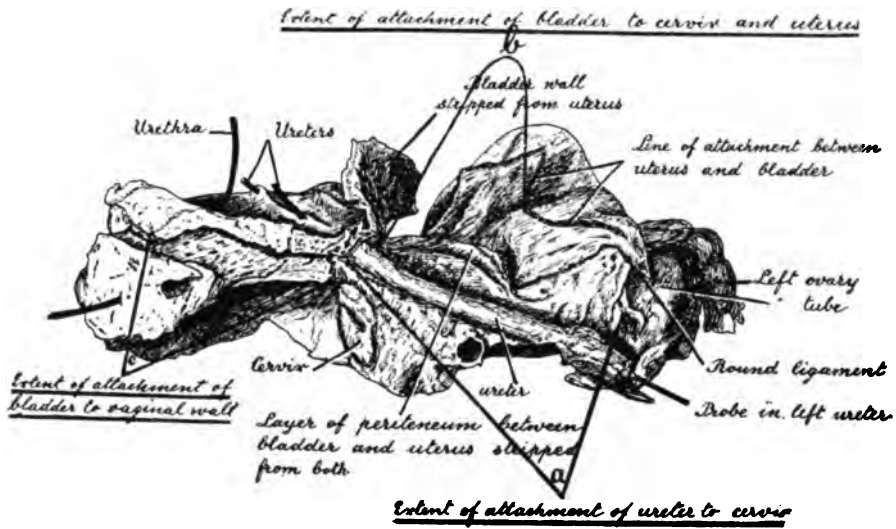


FIG. 1

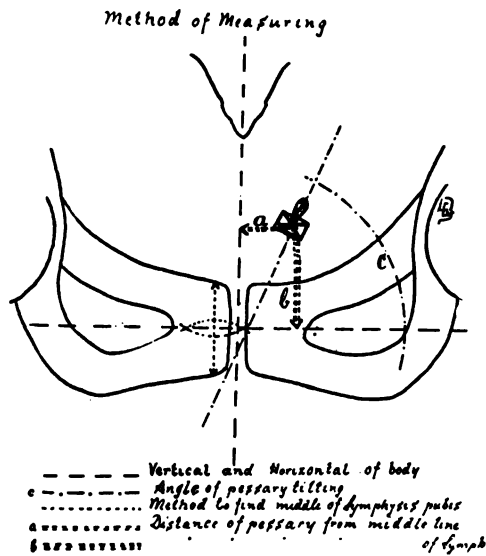


FIG. 2

Therefore, if the situation and the mobility of the uterine cervix can be accurately determined and recorded, the position of the uterus, the effect of the various positions of the same on the bladder and kidneys, and also the result of intra-abdominal pressure upon the urinary and other pelvic organs, would become a matter of scientific knowledge.

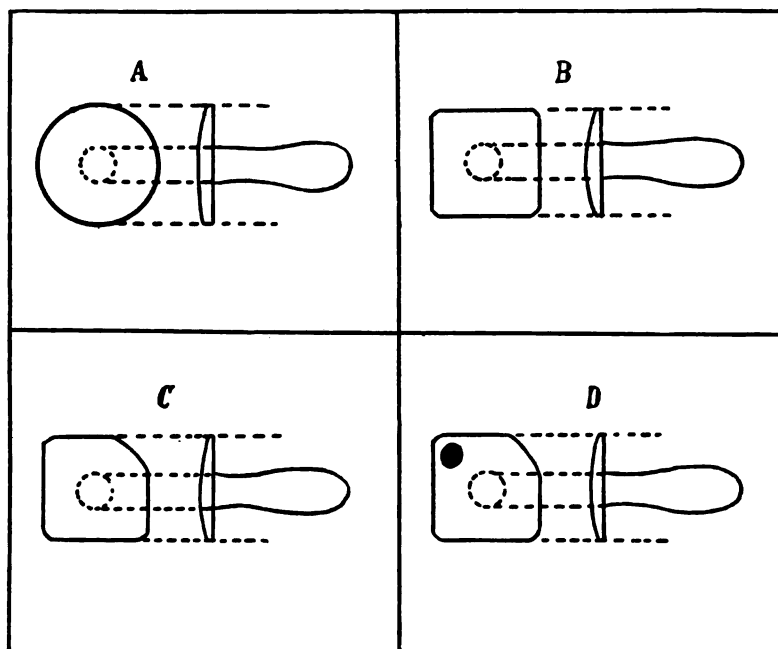


FIG. 3

Evolution of pessary

4th. The Röntgen rays show the position of metal in any part of the body. If a metal cervical pessary, therefore, can be so inserted in the cervix as not to interfere with the mobility of the uterus, we can readily obtain a picture of same in any position of that organ.

5th. By selecting fixed points on the body for measurement, the exact position and extent of the uterus can be ascertained and recorded.

The fixed points so selected are as follows (Fig. 2):

1. A horizontal line through the middle of the symphysis pubis.
2. A vertical line which intersects the horizontal at the median line of the body, *i.e.*, at the middle of the cartilage of the symphysis pubis.

The following measurements enable one to determine the exact position of the uterus:



FIG. 4

Old pessary in situ

1. The distance from the middle of the base of the pessary to the vertical line (Fig. 2, a).
2. The distance from the middle of the base of the pessary to the horizontal line (Fig. 2, b).
3. The angle of the shank of the pessary to a horizontal line drawn from the anterior to the posterior surface of the patient (Fig. 2, c).

X-RAY PESSARY TECHNIC

1. *Inserting Pessary*

Patient is placed in position for vaginal examination. The largest possible speculum is inserted, cervix brought into view, rendered aseptic and sterilized pessary inserted.

That the radiographic technic and the technic of interpreting the

radiographic findings may be recognized to be correct in principle, though as yet not wholly so in detail, we will briefly outline the evolution of the X-ray pessary.

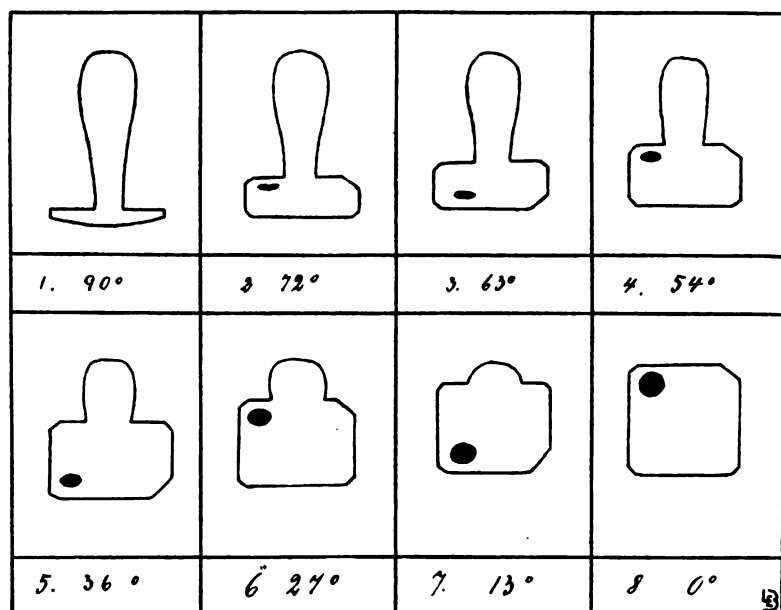


FIG. 5

X-ray shadow thrown by pessary at various angles (outside of body)

In our first enthusiasm we had not counted on the importance of shape. The shadow cast by an uninterrupted round base of a brass pessary (Fig. 3, a) did not allow the drawing of any conclusion as to whether the pessary had rotated or was pointing backward or forward, or both (Fig. 4).

To overcome this and facilitate measurement of angles, a square base was devised (Fig. 3, b), with one rounded corner (Fig. 3, c), the round corner making it possible to see on the X-ray plate whether the pessary had remained in the cervix as originally inserted or rotated. Furthermore, the pessary being composed throughout of the same metal, the base completely eclipsed the shank when extremely tilted anteriorly or posteriorly, making the

measurement of the angle at which the uterus was tilted an impossibility.

Later the important addition of a round opening cut in the base was made (Fig. 3, d), which affords another identification mark

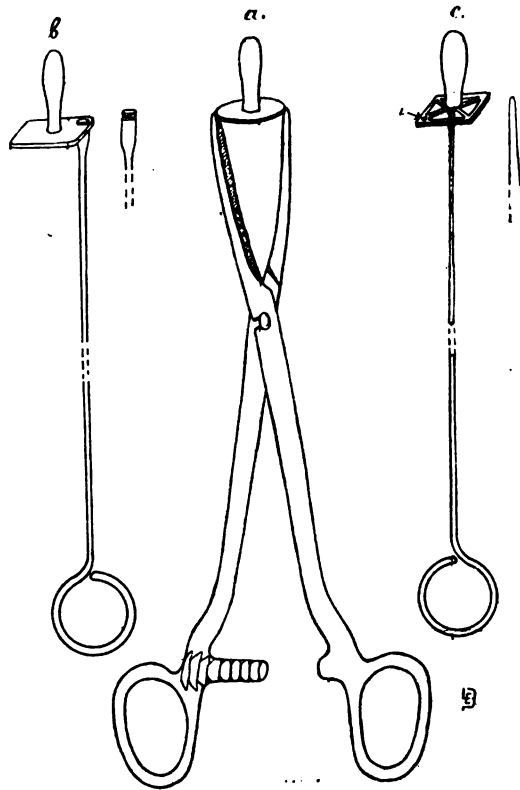


FIG. 6

Evolution (a-b-c) of holder for inserting X-ray pessary

as to the tilting of the pessary, by observing the size and shape of the hole on the X-ray plate (Fig. 5). Fig. 6 illustrates in the following order (a, b, c) the evolution and development of the holder for inserting the X-ray pessary.

The latest pessary (Fig. 7), though by no means perfect, still has many advantages, which are as follows:

The shank is composed of rubber and the base of very light metal, with the result that its light weight will not interfere with the position of the uterus, as that of the first pessary possibly did, since the difference in weight is 13.1 grams, or nearly $\frac{1}{2}$ ounce.

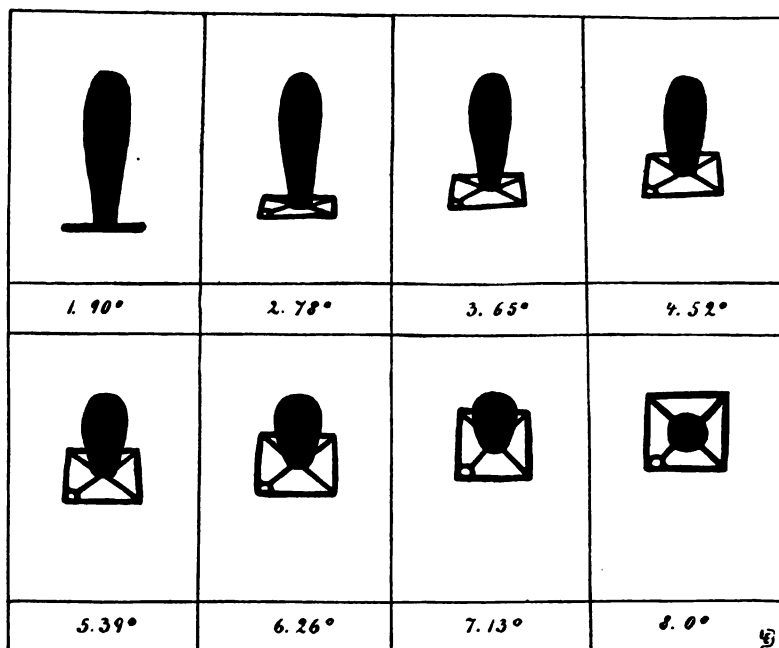


FIG. 7

New pessary with rubber stem—obliteration of angles showing tilting of uterus

In order to show any rotation or other movement of the uterus, the round hole in the base is always placed in one position. The position selected in our work is anterior and to the left side of the patient.

The four triangles free from metal enable one to see readily the cervical os, facilitate insertion of the instrument, and also permit one, knowing the position of the round hole in the base, to note the extent and the character of the uterine displacement by observing on the X-ray plate which triangle of the pessary is obscured.

2. X-Ray Technic

The series of pictures taken of normal and abnormal cases in various positions, Trendelenburg, standing, lying, without corsets, with corsets, with corsets laced tightly, etc., with many other points of evolution and technic, are omitted.

The fact exists, as already intimated, that to date we do not possess accurate knowledge as to the position of the normal non-pregnated uterus, the extent of its mobility, or its position under vari-



FIG. 8
Palpation

ous conditions, normal or abnormal. The only means of ascertaining the same is by palpation—a means that demands displacement from the normal, the overcoming of muscular resistance (Fig. 8), with entire failure to recognize intra-abdominal pressure as a factor. Moreover, it is a method that permits of ascertaining the position of the uterus in virtually but one position—the lithotomy position, not standing, with corsets, without corsets, etc.

Our present ocular knowledge is just as incomplete, for it is obtained at operation under general anesthesia or post-mortem. In

both these conditions two important factors governing the normal position of the uterus are entirely ignored—that is, intra-abdominal pressure and muscular tonicity.



FIG. 9
Patient lying, without corsets

But look at Fig. 9. There you see in a patient lying free from clothing the position of a normal uterus. In the next plate (Fig. 10) is shown the position the uterus assumed in the same patient standing, without corsets. Fig. 11 shows the position this uterus assumed when corsets were laced tightly, patient standing, waist compressed $2\frac{1}{4}$ inches.



FIG. 10
Patient standing, without corsets



FIG. 11
Patients standing, corsets laced tightly

In these three plates, the positions are different—so different as to be clearly apparent.

Here every physician reading this article sees—(think of it!)—has the same exact ocular perception of the position of a normal uterus in a living subject under these varying conditions. Have you ever before seen the position of a normal uterus in a living, conscious woman with abdominal wall intact? I think not!

Fig. 12 is a diagrammatic drawing made to readily illustrate the

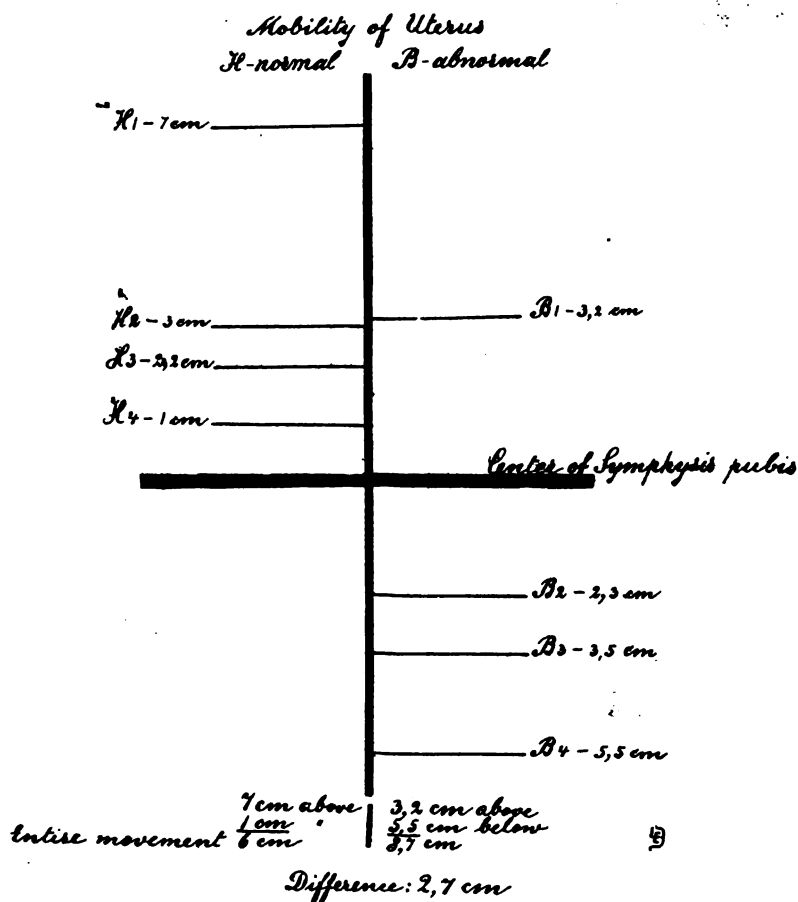


FIG. 12



FIG. 13
Normal patient, new pessary, bladder filled



FIG. 14
Patient lying, bladder empty, without corsets

difference in the range of motion in a virgin and multipara with lax intra-uterine ligaments and procidentia as shown by a series of X-ray plates taken with pessary in situ. The red line is the center of the symphysis pubis; the black horizontal lines on the left, marked "H" above same, show the position under varying conditions in a virgin; those on the right, marked "B," variation in a multipara. The difference in the range of motion and position is readily seen.



FIG. 15
Patient standing, corsets laced tightly



FIG. 16
Patient lying, without corsets, pessary and catheter in position

Is this knowledge as to the mobility of the uterus of practical value? I would say "Yes," in so far that whatever operation is employed to rectify uterine displacement, the principle governing it should be such as to permit the cervix a free range of motion between 1 cm. (.4 inch) and 7 cm. (2.8 inch) above the middle of the symphysis pubis. Again, the range of mobility obtained before operation will give information as to the tonicity of the uterine ligaments, therefore not only indicate the character of the opera-

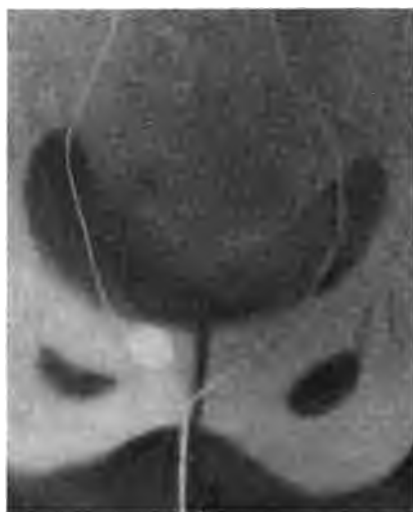


FIG. 17

Patient standing, without corsets, pessary and catheter in position

tion which is the best—whether suspension of the uterus, shortening of the ligaments, or both—but the exact amount of shortening of ligaments necessary, etc.

By means of the X-ray and the X-ray pessary, pathologic conditions in the genital and urinary apparatus in women may be seen as they exist before operation, and after operation the exact immediate and late post-operative results, and thus statistics of value as to the effect of various operations of suspension, etc., for procidentia, cystocele, etc., may be compiled. The exact character of

such pathologic condition and the results of operation, until the advent of this X-ray pessary, every one of us could surmise, but could not demonstrate.

It is my belief that this instrument—the X-ray pessary—will open a large and fertile field of study that will not only improve our knowledge of the genital and urinary diseases in women, but enable us to attain a much needed improvement in our surgical treatment.

The points taken are illustrated by the following:



FIG. 18

Patient lying, without corsets, bladder filled

Fig. 13, normal patient, new pessary, bladder filled.

Corroborates the statement that by observing the angle obliterated the position of the uterus can be defined. Here the posterior angle is partly effaced, demonstrating a tendency to retroflexion, due to fully distended bladder.

The next two illustrations (14 and 15), multipara with procidentia and cystocele, prove that we can ascertain the effects of corsets or constriction of the abdomen upon pelvic organs.

Fig. 14, bladder empty, patient lying, without corsets. Note the position of uterus. Fig. 15, patient standing. Note effects of old-fashioned, tightly laced corsets—uterus well depressed below symphysis.

The next two illustrations (16 and 17), multipara, procidentia

and cystocele, present two methods of X-ray diagnosis to show (1) how much the cystocele is due to relaxation of vaginal walls, (2) to the displacement of uterus. Fig. 16 exhibits the position the uterus assumes, patient lying, as illustrated by X-ray pessary, position of mouth of bladder as shown by X-ray catheter. Fig. 17 shows position of uterus, also of mouth of bladder, when the patient is standing. The exact difference is plainly discernible.



FIG. 19

Patient standing, without corsets, bladder filled



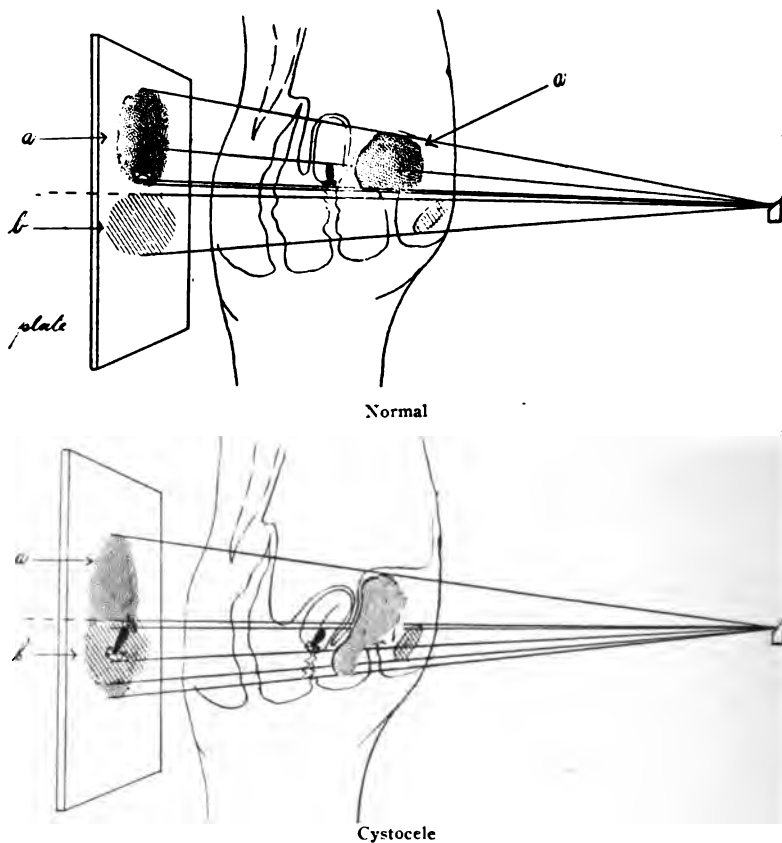
FIG. 20

Patient standing, without corsets, bearing down

Figs. 18, 19 and 20 show the position of uterus in a nullipara, slight procidentia, no symptoms, bladder filled with two per cent silver iodide. Fig. 18, patient lying. Note the bladder is above the symphysis pubis, as is also the cervix uteri. Fig. 19, patient standing, exhibits bladder sufficiently below symphysis to permit of a diagnosis of cystocele, but the causative factor is the more pronounced procidentia, as evidenced by the position of pessary, which is not only considerably below center of symphysis pubis, but also much below bladder. Fig. 20 is taken with patient standing, but bearing down. Note there is practically no difference of uterus between this and Fig. 19, showing the value of an intact vaginal wall as a uterine support.

Fig. 21 is a diagrammatic drawing we employ in our case histories in noting position of uterus and bladder.

Though this means of diagnosis is in its experimental stage, nevertheless when a patient comes who has urinary symptoms, frequency, etc., with various diagnoses as to degree and form of malposition of the uterus (as varied as the number of physicians con-



Cystocele

FIG. 21

Drawing for case histories. X-ray—(a) shadow of bladder; (b) shadow of symphysis. Normal—bladder and pessary above symphysis. Cystocele—bladder and pessary below center of symphysis

sulted), one can make the diagnosis, demonstrate the findings in black and white to the patient on the X-ray plate; then is the practical value of the X-ray pessary evident. It makes the diagnosis an exact science—which the public is not slow to appreciate.

URINALYSIS AND THE GENERAL PRACTITIONER

By ROBERT KILDUFFE, JR.

Director, Pathological Laboratory, Chester Hospital
Chester, Pa.

This is an age of scientific medicine, or, as might be said, of etiologic medicine, having as its keystone accuracy in diagnosis, pointing the way to accuracy in treatment; and each year the energies and the skill of the theorist, the investigator, and the clinician are bent toward the development and perfection of diagnosis as an exact science.

It is not my intention to even touch upon the many advances in diagnostic technic which belong fairly and entirely to the laboratory man, but rather to confine myself to those within the reach of the general practitioner—and not to all of them, for if he but realized it, his activities in this field are far from restricted—but to one alone—urinalysis.

Urinalysis is one of the most useful, and most neglected, of diagnostic procedures at the service of the general practitioner. This is a statement which can be made without fear of contradiction, for the method of urinalysis as practised by the average practitioner consists, as a rule, of one of two ancient and classic procedures. The first of these is to allow the bottle to stand until obtrusively ammoniacal and then drop it into the waste-basket, and the second, to employ the "sink test," which has, at least, the value of rapidity and cleanliness.

The explanation, I believe, lies mainly in the multiplicity of tests with which the various text books and manuals abound, and the amount of apparatus necessary for their performance—to say nothing of the skill and experience demanded by their technic and the interpretation of results.

It is true that there are laboratories galore from which voluminous reports may be had, and reports of great value to those who can interpret them, but either the patient cannot or will not pay for the examination, or they are so exceedingly complete as to still further becloud the diagnosis of a doubtful case, and again the urinalysis is neglected or its results pooh-poohed.

I would like at this point to say a few words in regard to the inter-

pretation of a urinalysis and of laboratory reports in general. Let it be borne in mind that anyone, without exception, can be taught to put this and that together in a test tube, or to make this or that stain, or prepare, section, and stain a tissue, and successfully carry the performance to its ultimate conclusion—and the result is simply a completed test. The interpretation of the result, the meaning of that test with regard to that definite case, is a very different matter entirely and requires an altogether different person. It is the man behind the test who makes it worth doing. The man who thinks that surgery, that drugs, that electricity, or massage *alone* are cure-alls, is close kin to him who thinks that the art of diagnosis rests alone with the laboratory; that all he has to do is to drop a test in the slot and draw out a neatly printed diagnostic card in return; and he wears the same blue spectacles and travels in the same rut with the man who looks upon the laboratory as a fanciful and ornamental way of wasting time.

Let us not forget that there is not a single advance in either medicine or surgery, in pathology, hygiene, or sanitation, which does not depend upon the coöperation of the laboratory worker, upon his ingenuity, his skill, and above all, his patience. Remembering that, let us treat him fairly as a brother consultant and as such, entitled to his due.

We would not call in a surgeon and, depriving him of the history and physical examination, say—"Here is a twisted knee—what is the matter with it?" Or a physician and say—"Here is a man with a cough—what has he got?" But it is quite the proper procedure to get a specimen of urine, or of blood, and because we find albumin or an anemia, to expect an answer to the question, "What has he got?" Or, in spite of the fact that the same cells which in the skin of the hand are normal, in the breast spell carcinoma, to cut a piece squarely from the center of a tumor mass, to put it into the first solution at hand, or even, as I have seen, none at all, and without any history, without any knowledge of the symptoms, without any idea of where the specimen came from, to ask the pathologist, "What has he got?" We all know the mere presence of albumin in the urine does not make a diagnosis of Bright's disease, that there are many factors which must enter into the interpretation of its presence and determine its diagnostic value—in a word, that it is the

interpretation of the test that counts; but if we cannot interpret it, do we consult the books, or better still, give the laboratory man the same facts we possess in regard to the patient and ask his interpretation as we would any other consultant? We do not. We either disregard the analysis or take it as another evidence of the uselessness of laboratory examinations in a doubtful case.

Do not think that you can always make the diagnosis by the laboratory alone or without it; the diagnosis is always the sum total of the history, the physical examination and the laboratory test, and not infrequently it is the last which points the way.

Returning from this digression, it is my purpose to call attention briefly to the value of a routine analysis in all cases, and what is more important, to the small number and extreme simplicity of certain tests which are as easily made and as easily interpreted by the general practitioner.

It may be of interest to note, briefly, the more salient points in the history and evolution of this most ancient procedure—the diagnosis of pathological conditions by the examination of the urine.

In the British Museum are fragments of a Syllabarium or dictionary of words relating to parts of the body, among which are signs and words relating to urine which show that as early as 4000 B. C. the Babylonian physicians had noted changes in its color and composition.

The Hindu physicians, in their earliest records, make frequent and lengthy reference to the examination and character of the urine, and under each of the twenty morbid conditions which constituted their pathology are grouped various classifications of the urinary changes. It is interesting to note that they describe a "honey urine" which would attract ants (which fact they used as a test), thus evidencing a knowledge of diabetes, and, moreover, they noted its relation to carbuncle and other skin affections, and commented upon the hereditary and congenital forms of this disease.

As early as 100 B. C. Charaka describes enlarged prostate as follows: "Deranged wind between the bladder and rectum produces a hard ball like a stone which is the cause of suppression of urine and causes much pain and swelling of the bladder."

In the early Greek era, 400 B. C., we find Hippocrates frequently urging the importance of the examination of the urine in prog-

nosis, and he writes at length on its characteristics in various conditions, many of his comments, though too lengthy to be included here, being extremely shrewd and interesting.

Galen, in 130 A. D., largely follows the teachings of Hippocrates, as does Paulus Aequinata, a famous physician of the seventh century who wrote extensively on the subject.

In 610 A. D. Theophilus makes the first reference to the application of heat and alludes to the cloudiness sometimes arising.

Ismail of Jurgani, a Persian physician of the eleventh century, gives detailed directions as to the manner in which a urinalysis shall be made, recommends the collection of a twenty-four hours' quantity, and enumerates the points to be noted—color, consistency, transparency, quantity, sediment, odor, and froth.

Such was the dignity of urinalysis that Johannes Actuarius, who died about 1283, held the position of uroscopist to the Byzantine court and wrote an extensive work on the subject, being the first to recommend the use of a graduated glass to measure the deposit.

In the early Middle Ages inspection of the urine came second to the pulse, and a chronicler of the time shows us that there are some medical procedures of that age still in vogue when he says that, after concluding his examination, and before retiring, the physician usually promised that, with God's help, the patient would recover, but "he would take care to inform the friends of the patient that the illness is a serious one, so that, should the patient get well, the merit may be due to him, or should the result be fatal, the friends of the deceased are there to witness that he had noted the serious nature of the disease from the first."

Probably the earliest book published in the English language on urinalysis is entitled "The Judycyall of Uryns," and is said to have been printed in 1512.

Another such manual was published in 1540, and still another, "The Urynal of Physik," in 1548 by Mayster Robert Recorde, which is very detailed in its directions.

Up to this time urinalysis consisted almost entirely of inspection, the diagnosis and prognosis being made on its appearance, and it was not until 1655 that the first inquiry was made into its composition by Van Helmont, who also devised a method of determining its weight.

We must not forget here the influence of that peculiar combination of scientist and quack, Paracelsus, who, referring all diseases to the chemicals of which, he said, the body was formed, namely, mercury, sulphur and salt—mercury belonging to the lower limbs—distilled the urine to determine which element was in excess, thus determining the location and nature of the disease.

In 1674 Thos. Willis made the first observation of the sweet taste of diabetic urine, thus paving the way for the distinction between diabetes insipidus and glycosuria.

Considering the importance given to the examination of the urine and the impression made upon the imagination of the ignorant, it is not surprising that quack urologists of every description abounded, who, gathering their auditors in the market-place, without ever seeing the patient, by inspection of the urine did not hesitate to extend their divinatory powers to all kinds of predictions beyond the range of medicine.

It became a common practice for apothecaries to collect specimens and take them to physicians, who by inspection made a diagnosis, leaving the treatment to the man of drugs, and to such an extent did this go that it later was forbidden by law.

Shakespeare alludes to the examination of urine in King Henry IV when Sir John Falstaff asks his page: "Sirrah, what says the doctor to my water?" The page replies: "He says, sir, the water itself is a good, healthy water, but for the party that owns it, he might have more diseases than he knows for."

The first estimation of specific gravity was made about 1712 by Boorhaave, a Dutch physician, and inaugurated an era of scientific urinalysis; in 1720 he also discovered urea, and in 1772 Mathew Dobson, of Liverpool, demonstrated sugar in diabetic urine.

Bile was discovered in 1789 by Cruikshank; a test for albumin in 1801 by Jaroll, and in 1841 the first test for sugar was described by Trommer, the test which now, modified, bears Fehling's name.

From this on to our own day the gradual evolution of modern laboratory methods is familiar to us all. Unfortunately, in some respects, the subject has become so complex that the average practitioner has become confused in its mazes, and not infrequently, at first puzzled, later becomes dubious and ends by disregarding the matter almost entirely.

For the man who makes his own examinations—and there are a few who cannot—there are two valuable principles to remember:

1. It is better to know one test well; to understand its fallacies and its limitations, its interpretation, than to have a smattering knowledge of many and a thorough understanding of none.

2. It is not the test but its interpretation that counts.

With these facts in mind, we come to our tests, and I hope to present a short and simple routine method of urinalysis which will help to clear up many a knotty point and indicate the case in which the more detailed methods of the laboratory man are called for.

I shall not go into details as to the interpretation of the tests, as that depends upon the man and his reading, but merely give the tests themselves.

1. *Quantity*.—This is a point of marked diagnostic value, but, even though necessitating no extra labor on the part of the physician, is, nevertheless, except in the exceptional case, almost entirely neglected.

It is almost needless for me to emphasize the almost utter uselessness of the perfunctory examination of one single specimen passed simply for that examination.

It is true that cases of glycosuria and albuminuria in which the pathological elements are constantly present in large amounts are readily detected by such an examination, but these are not the cases in which a urinalysis has its greatest value; the diagnosis, and to some extent, the progress of the case, are clear from the symptoms.

It is in the border-line case that we need, and look for help, and when we remember the changes in the character of the urine and its constituents which may be due to factors of diet and habits of life; when we remember that there may be marked fluctuation of sugar and albumin, or of pus, blood, bile, etc., at different periods during twenty-four hours; that casts may occur in showers and be absent in the interim, the fallacy of any conclusions drawn from the examination, no matter how careful, of a single specimen are at once apparent.

I would make a strong plea, then, for the examination of a specimen from the mixed twenty-four hours' quantity. Its collection is an easy matter and involves but very simple directions to the patient:

1. To empty the bladder at some definite time.

2. To keep every portion of urine passed from that time to the corresponding hour next day, including what the bladder then contains.

3. To measure this quantity and bring two ounces for examination.

The addition of 5 to 10 grains of boric acid will effectually prevent decomposition and will not interfere with any tests.

If we bear in mind that the daily output is influenced by the habits of the patient with regard to the amount of fluid ingested, the nature and quantity of food, the blood pressure, temperature and amount of exercise, and apply these factors to the individual, we are often at once placed in possession of valuable data as to that individual's renal functional ability.

The normal twenty-four hours' quantity in the United States may arbitrarily be placed at 1,000 to 1,200 cc. in the male, and 900 to 1,100 cc. in the female.

2. *Inspection.*—This is another neglected factor. Much may often be learned by merely looking at a specimen. Its color is of interest, often an indication of the presence of blood, pus, or bile, or certain drugs, as, for example, the smoky appearance of carbolic urine, the yellow of santonin, the red of logwood, etc.

In this connection it is well to remember that in cases of hysteria and malingering dyes may be purposely added. Remember, too, that the greater the density the darker the color, and *vice versa*, is a general rule of value; that an acid urine and a febrile urine are generally darker, and that a very pale urine, though generally indicating an excessive ingestion of water, may indicate chronic interstitial nephritis, diabetes (either variety), hysteria, or the various anemias.

3. *Specific Gravity.*—This is only of value, and can only convey information when taken from the twenty-four hours' quantity. Under such conditions it is, in a general way, an index of the metabolic processes of the body, increasing as the solids increase, the amount of urine remaining the same. The normal range of variation is from 1.015 to 1.025, and by multiplying the last two decimal points by two we get the solids for 1,000 cc., from which the twenty-four hours' amount is readily calculated.

We come now to the actual tests, and it is my hope to present them in such simple form that their routine use will be stimulated.

4. *Albumin*.—For general use the best and most dependable test is the nitric acid contact test, which should be performed in every detail exactly as follows:

1. Do not make the test in a test tube.
2. Do not stratify the urine on the acid.
3. Into a small conical glass (medicine glass) pour 10 to 20 cc. of urine and, inclining the glass, allow 6 to 10 cc. of nitric acid to flow down the side, forming a distinct zone below the urine.
4. Allow the test to stand five minutes.

In almost every specimen, in the clear urine above the point of contact a fine white ring will appear after standing. This is not albumin and although information on this point is not exact, is generally referred to urates and has no clinical significance.

The white albumin ring appears almost at the point of contact and, to some extent, varies in amount and intensity with the amount of albumin present, and if much is present the cloudiness will gradually extend upwards into the supernatant urine. If bile is present, beneath the albumin disc the characteristic green ring will be seen; urea, if present in more than 25 grams to the liter, will cause a hoarfrost of urea nitrate on the sides of the glass.

A transparent ring, varying in color from pink to brick red, seen in the body of the urine is referable to normal urinary pigment.

Indican appears as a violet ring above the so-called urate ring.

Remembering that certain resinous bodies may be precipitated from turpentine, balsam of tolu, capaiba, etc., which cloudiness, if shaken with alcohol, at once disappears, it is evident that this simple test is of incalculable value and its routine and general adoption cannot be too strongly urged.

In the exceptional case, where there may be some doubt as to the reaction, I have made use of the following simple but effective modification of technic: Into a test tube three-quarters full of urine introduce a 2 cc. pipette charged with nitric acid. If, with the tube held against a dark background, the acid is released in spurts, albumin appears like little clouds of smoke from a gun whirling up into the clear urine above which acts as a control.

Bearing in mind the albuminuria which may follow the exces-

sive ingestion of albuminous foods over the assimilation limit, and excluding that due to blood, pus, etc., the interpretation of the test is clear, and it is to be noted that there is a growing tendency to cast aside so-called "physiological albuminuria" and regard the presence of albumin in every case as a pathological phenomenon.

Quantitative Estimation.—This is equally simple, the only apparatus required being the familiar Esbach tube. For seven years I have used, and would advise Tsuchiya's reagent as having several advantages over Esbach's in that it is more stable, more accurate in not precipitating as many extraneous substances, and does not stain. It can be readily made by any druggist from the following formula: Phosphotungstic Acid, 1.5 gram; Alcohol 95%, 95 cc.; Conc. HCl, 5 cc.

To make the test pour urine into the tube to the mark U, add the reagent to the mark R, invert several times and set aside for 8 to 12 hours. The graduations of the tube refer to grams per liter. The reaction of the specimen must be acid.

Sugar.—I have not used Fehling's solution for this test for some years, preferring to use Benedict's reagent, which has the following formula: Copper sulphate, 17.3 grams; Sodium citrate, 173.0 grams; Sodium carbonate, 100.0 grams; Water, 1,000 cc. Benedict's reagent is based upon the same principles as Fehling's solution—the reduction of copper sulphate—and has the same end reaction; a yellow or red precipitates in the presence of sugar; but the solution has several advantages: (a) it is stable, (b) there is only one solution, (c) it is slightly more delicate.

The technic is extremely simple. The solution is diluted and used exactly as in Fehling's test, or the simple modification of technic described below may be used with decided increase in the delicacy of the test and provides an effective safeguard against some of the fallacies:

1. Acidify and boil a few cubic centimeters of urine in a test tube, thus precipitating any albumin present.
2. Boil a few cubic centimeters of the diluted reagent in another test tube.
3. Place a small filter with moistened filter paper in the reagent tube with the beveled edge of the funnel against the side of the tube.

4. Pour the boiling urine into the filter.

As the hot urine runs through the filter it stratifies upon the hot reagent and, in the presence of sugar, a beautiful ring reaction will be obtained.

By this technic the delicacy of the test is decidedly increased and, moreover, fallacious reactions due to albumin and the reduction of copper by creatinin, etc., are effectually eliminated.

Bear in mind the fallacies of any test depending upon the reduction of copper sulphate. Not only certain drugs such as benzoic acid, salicylic acid, glycerin, chloral, sulphonal, etc., but certain other substances normally present in the urine may cause reduction. These, however, even if the above technic is not used, may be disregarded if the precaution is taken not to boil the test after the addition of the urine.

The quantitative estimation of sugar, which is extremely important, as a rule, requires more skill, practice and apparatus than are generally at the disposal of the busy practitioner, and I pass it over, suggesting only to those who desire to do it the use of Purdy's solution as having the advantages of a clear and unmistakable end reaction, thus necessitating only one estimation.

There are many other tests of simple technic, but it is better to do a few tests always than many only once in a while, and I pass them over. The examination of the sediment, while of extreme value as the only reliable test for pus and blood and the only test for casts, and a means of obtaining much and varied information, I leave to the laboratory man, for it lies in his domain, as it requires not only a microscope but also what is more important, the man behind the 'scope to interpret what the picture means.

To him also belongs the phenolsulphonephthalein functional test, which should be done more frequently in practice, for, not only harmless to the patient and simple in technic, it can give us much information as to changes and derangements of renal function even before the microscope or the test tube can detect any variation from the normal.

Progress of Diagnosis and Prognosis

GENERAL METHODS OF EXAMINATION—SYSTEMIC AFFECTIONS—DISORDERS OF GENERAL METABOLISM

Alveolar Carbon Dioxid Determinations—E. P. POULTON, Brit. Med. Jour., Sept., 11, 1915.

If the urine in a case of diabetes gives a negative result with the nitroprussid test, it is useless to make an alveolar carbon-dioxid determination, for it will always fall within normal limits. The alveolar carbon-dioxid determination is a sure guide as an index of the degree of acidosis in those cases in which the urine contains acetone bodies. In such cases it is usually impossible to determine the degree of severity by means of the ferric chlorid or nitroprussid tests alone. In cases in which the alveolar carbon-dioxid pressure is lower than normal, care must be taken to prevent the onset of coma. A value of 2 per cent. means that coma may supervene within 24 hours. A value of 3 or 4 per cent. is less dangerous. SACHS.

Acidosis in Diabetes Mellitus—A. P. BEDDARD, M. S. PEMBRY and E. J. SPRIGGS, Brit. Med. Jour., Sept. 11, 1915.

The analysis of the carbon dioxid in the alveolar air spaces of the lungs affords an index of the degree of acidosis and a guide in treatment and prognosis. In cases of diabetes, there exists a relation between the alkalinity of the serum and the amount of carbon dioxid in the venous blood; the two rise and fall together without being actually parallel. SACHS.

Occurrence of Arsenic in the Female Organism—V. FROMMER, Archiv f. Gynäkologie, Vol. CIII, No. 2, 1915.

In pregnant animals, to whom arsenic was administered, the transmission of arsenic from the mother-animal to the placenta and fetus could be demonstrated in every instance. In the human organism the presence of arsenic is very likely due to alimentation. This is of import in forensic medicine and in biochemical respect. In a number of human organisms arsenic in variable amounts could be demonstrated. Symptoms of gravidity and eclampsia are in many respects analogous to such of arsenic, phosphorous, etc.

MILL.

Staining Cells in the Cerebrospinal Fluid—B. LEMCHEN, Med. Rec., Sept. 11, 1915.

In staining cells in the cerebrospinal fluid, author uses an equal amount of two solutions. No. 1 consists of an equal amount of

benzidin in glacial acetic acid and No. 2 consists of hydrogen peroxid. By means of a white blood pipet, he draws up the stain to 0.5 and then the spinal fluid up to 11. This mixture is then put on a counting chamber. The red blood cells are stained blue; the polymorphus cells are a light yellow while the nucleus is made visible by having blue granules in it. In the lymphocytes the periphery is a ring of dark blue, while the middle of the cell is unstained, except for some blue granules. The periphery of the plasma cells is a dark blue, almost black, while the middle of the cell is clear. SACHS.

The Origin of Local Eosinophile Cells—B. PHOTAKIS, *Zeitschr. f. experimentelle Pathologie u. Therapie*, Vol. XVII, No. 2.

Eosinophile cells of the blood and bone marrow flow to such regions where specific, attracting substances are accumulated. If the latter accumulate in a certain localized area they attract a number of eosinophile leukocytes from the blood and bone marrow, giving rise thereby to a local eosinophilia. WESTERN.

Aleukia Hemorrhagica—E. FRANK, *Berliner klin. Wochenschr.*, Sept. 13, 1915.

The aplastic anemia, which should more definitely be denominated aleukia hemorrhagica, is not a primary hemolytic erythrotoxicosis with secondary absence of regeneration, but a primary leukomyelotoxicosis with secondary anemia. The latter bears partially a post-hemorrhagic, partially a myelophthisic character. Aleukia hemorrhagica is a well-circumscribed affection, and not a special form of certain secondary anemias or of the Birmer-Ehrlich anemia. MILL.

A Urobilin Test of the Urine and the Feces—A. EDELMANN, *Wiener klin. Wochenschr.*, Sept. 9, 1915.

Author describes a modification of Schmidt's test. Two reagents are necessary: (A) a concentrated alcoholic solution of mercury bichlorid, (B) a 10% clearly filtered alcoholic solution of zinc chlorid. Besides these amyl alcohol is essential. About 10 cc. urine is added to half its volume of A. To this is added amyl alcohol and the entire mixture well shaken. To the alcohol layer, which forms rapidly on top of the mixture, a few cubic centimeters of B. is added. In case larger amounts of urobilin are contained in the urine, the alcohol assumes a rose-red coloration; addition of the zinc chlorid solution calls forth a beautiful green fluorescence. Even small amounts of urobilin are recognizable by this test by employing after the reaction has been displayed a convex lense (pocket lantern) by which a green cone of light is produced in the liquid. The fluorescence reactions only ensue when the media are clear. In the urine the reaction is displayed in about one, in the stools in about two minutes. MILL.

Diagnosis of Metastatic Tumors of the Bone-Marrow from the Blood

Examination—J. v. ROZNOWSKI, *Zeitschr. f. klin. Medizin*, Vol. LXXXI, Nos. 5 and 6.

The appearance in large numbers of myelocytes in the blood of cachectic persons points with great probability to the presence of bone-marrow metastases of a malignant tumor. This is especially the case when the malignant tumor is demonstrable, and when there exists a grave secondary anemia with very large amounts of normoblasts, occasionally also megaloblasts, with or without synchronous leukocytosis.

WESTERN.

Pathology of the "Cold"—Aufrecht, *Deutsches Archiv f. klin. Medizin*, Vol. CXVII, No. 6.

"A cold" is due to coagulation of fibrin in the blood current. The cause of this coagulation is due to the injury of the leukocytes circulating through the vessels of the cooled-off portions of the body. The consequence of the coagulation is a hemorrhage in the peripheral blood vessels clogged by the coagulated fibrin. These changes are not engendered by bacteria. The fibrin coagulation, however, and its causative factor are pathological processes.

WESTERN.

The Influence of Muscular Activity upon the Blood Sugar—W. v. MORACZEWSKI, *Berliner klin. Wochenschr.*, Oct. 4, 1915.

The blood sugar is increased after almost every diet when work is performed. This is especially the case in a person tending to glycosuria. The diabetic shows a distinct increase in blood sugar also after the ingestion of a fatty diet. The muscular activity test is therefore equal in value to the ingestion of sugar to determine whether and in what degree an organism tends to glycosuria.

MILL.

Pituitary Gland in Diabetes Mellitus and Disorders of the Glands of Internal Secretions—H. J. B. FRY, *Quart. Jour. Med.* (London), July, 1915.

Definite histological changes occur in the anterior lobe of the pituitary in cases of diabetes in the form of adenomatous masses of eosinophilic cells, colloid invasion of the anterior lobe, and areas of cellular degeneration. In acute pancreatitis and carcinoma of the pancreas, changes in the pituitary are absent or slight. No histological changes were observed in a case of Addison's disease or in a case of status thymo-lymphaticus. Increase in the weight of the pituitary occurs in myxedema due to increase of the connective tissue elements and hyperplasia of the chief cells. In goiter there is hyperplasia of the chromophile cells, especially of the eosinophilic granular cells and increase of colloid in the interglandular cleft.

SACHS.

INFECTIOUS DISEASES

Examination of Tuberculous-Meningeal Spinal Fluid by Means of the Ninhydrin Reaction—V. KAFKA, *Münchener med. Wochenschr.*, Oct. 5, 1915.

The ninhydrin test with the spinal fluid, as employed by Nobel, cannot be employed in the differential diagnosis between the tuberculous and other forms of acute meningitis. In affections of the central nervous system in which there is an increase of spinal liquid albumin, the test should only be utilized with very great caution. The differential diagnosis between these affections may be accomplished by dialyzation of the spinal liquor with distilled water and employing the ninhydrin test with the dialysate. MILL.

Tubercle Bacilli in the Blood of Tuberculous Patients—L. KESSEL, *Am. Jour. Med. Sci.*, Sept., 1915.

Author pursued the following methods: 1. The blood taken from patients with advanced pulmonary tuberculosis was inoculated directly into the peritoneal cavities of guinea-pigs. 2. The blood from some of these patients was treated and examined microscopically. 3. The blood from tuberculous patients who had previously received an injection of tuberculin was inoculated intraperitoneally into guinea-pigs. 4. The blood was withdrawn from tuberculous patients who had previously received a tuberculin injection, and after removal of the serum intraperitoneal injections were made into guinea-pigs. 5. The blood, after removal of the serum, was planted upon culture tubes of gentian-violet media. The following summary of his observations is given by author: 1. Blood withdrawn from 38 patients was inoculated intraperitoneally into guinea-pigs. Autopsies and microscopic sections two to three months later failed to reveal any evidence of tuberculosis. 2. Microscopic examination of the blood withdrawn from 10 patients yielded negative results. The many sources of error in the microscopic examination of blood for tubercle bacilli have been pointed out. 3. The blood of 7 patients previously subjected to a tuberculin injection was inoculated intraperitoneally into guinea-pigs. Autopsies and microscopic sections failed to reveal any evidence of tuberculosis. 4. In 3 patients who had previously received a tuberculin injection, blood was withdrawn and after removal of the serum was inoculated intraperitoneally into guinea-pigs. One of the pigs developed an extensive tuberculosis. 5. An attempt made to grow the tubercle bacilli directly from the blood proved unsuccessful. The negative results yielded by these 47 cases do not prove conclusively that tubercle bacilli are never present in the circulation, but they strongly suggest that a bacillemia, such as is present in other infectious diseases, is at least uncommon in pulmonary tuberculosis even in advanced stages of the disease. It may be that from time to time tubercle bacilli

are washed into the circulation from a pulmonary focus, and that they rapidly disappear from the blood. That such a rapid departure from the blood does occur, has been demonstrated in the case of rabbits in whom tubercle bacilli could no longer be recovered from the blood thirty minutes after their intravenous inoculation (Heymann and Otto). When the blood of 10 patients previously subjected to a tuberculin injection was inoculated into guinea-pigs, one of the animals presented a generalized tuberculosis at autopsy. It would be unwise to draw conclusions from one positive result, but the 9 negative results coincide with our daily clinical experience, for if a therapeutic tuberculin injection could cause virulent tubercle bacilli to appear in the circulation the development of acute miliary tuberculosis would be a common occurrence. Nevertheless, the question of a possible mobilization of tubercle bacilli following diagnostic and therapeutic tuberculin inoculations deserves careful investigation.

SACHS.

Percussion and the Diagnosis of Apical Tuberculosis—A. SCHNEIDER, *Deutsche med. Wochenschr.*, Aug. 12, 1915.

Tuberculosis of the pulmonary apices is, as a rule, physically first demonstrable by auscultation. Negative percussatory findings do not exclude a tuberculous affection of the apices. In spite of such negative findings there may be present an extensive moist tuberculous catarrh. Tuberculosis of the pulmonary apices is in most cases only demonstrable by percussion when the Röntgen examination is already positive.

MILL.

Early Diagnosis of Pulmonary Tuberculosis—E. G. GLOVER, *Quart. Jour. Med.* (London), July, 1915.

Sixty-two per cent. of cases coming under observation as supposed cases of early phthisis, but with no bacilli in the sputum, proved to be negative. Nevertheless such negative cases may present clinical signs of impairment of percussion-note, breath sounds and resonance, at, at least, one apex. Moist sounds in such cases are almost invariably absent. Therefore it is not justifiable to diagnose active tuberculosis on the strength only of impairment of the percussion note at an apex. Active disease confined to one apex with a repeatedly negative sputum is not common. Where moist sounds are present, further investigation is needed to exclude or confirm the presence of active disease. When this investigation takes place, the form of the complement-fixation reaction along with the estimation of the opsonic index may make a final diagnosis without recourse to test injections of tuberculin.

SACHS.

Graves' Disease and Tuberculosis—SCHINZINGER, *Beiträge z. Klinik d. Tuberkulose*, Vol. XXXIII, Nos. 1 to 3.

Basedow disease occurs not more frequently in the presence of pulmonary tuberculosis than in that of other diseases. It is dubious

whether or not the symptoms of the first stage of pulmonary tuberculosis, as cardiac palpitation for instance, have anything to do with Basedow disease.

FRY.

Tests for Syphilis—H. C. BROWN and K. R. K. IYENGA, *Indian Jour. Med. Research*, July, 1915.

The first test for syphilis which the authors have devised is dependent on the fact that when an aqueous solution of platinum chlorid is added to an alcoholic extract of normal serum, a much heavier precipitate is produced than in the cases of an extract of syphilitic serum. The results of this test parallel those of the Wassermann reaction and Flemming's modification. The second test is a natural consequence of the first test. Since one of the properties of lecithin differentiates a syphilitic from a normal serum, the other should also do. The capacity for activating cobra venom is different in the two serums. Fifty-five serums tested bore out this conclusion.

SACHS.

Splenic Enlargement in Early Syphilis—U. J. WILE and J. A. ELLIOT, *Am. Jour. Med. Sci.*, Oct., 1915.

The spleen was definitely enlarged in 36 out of 100 cases of early syphilis studied by authors. It was hard and firm in 17 cases, tender in 6 and soft in 3. Twenty-seven of the 36 cases showed impairment of health, but it must also be noted that there was impairment of health in 22 per cent. of those cases in which the spleen was not enlarged.

SACHS.

Congenital Syphilis in Prematurely-Born Infants—A. REICHE, *Zeitschr. f. Kinderheilkunde*, Vol. XII, No. 6, 1915.

Among prematurely-born infants there are comparatively more affected with syphilis than among those born at term. Syphilitic premature births exhibit a high mortality, especially when the luetic manifestations appear early, and when the mother has had a number of premature births or abortions.

MILL.

Gonorrhea: Complement-Fixation Test—A. A. UHLE and W. H. MACKENNEY, *N. Y. Med. Jour.*, Oct. 9, 1915.

The blood of 141 individuals was tested by each of 4 competent serologists for complement-fixation. Fifteen of the 141 were normal controls. One laboratory reported negative in all of these cases, while the other three serologists gave positive reports in from 6.6 to 13.3 per cent. In 37 cases, all of which, while suffering from other diseases, denied ever having had gonorrhea and presented no clinical evidence of the disease, three serologists reported positive findings in from 13.5 per cent. to 35.1 per cent., and one laboratory found all the bloods to be negative. Of 18 cases clinically cured for over five years, all of the laboratories agreed on a negative report in 8

cases. In 11 cases of acute gonorrhea of less than 4 months' duration, all of the laboratories agreed in a negative report in 3 cases.

SACHS.

Degeneration Forms of Gonococci—ASCH and ADLER, *Münchener med. Wochenschr.*, Sept. 28, 1915.

The bacteriological diagnosis of acute gonorrhea is comparatively easy. This is by no means the case if the urethritis has attained a certain degree of chronicity. Here a vast number of other bacteria are found besides the gonococci. This fact makes the diagnosis more difficult. Authors demonstrated that in most cases the gonococci are only Gram-negative in the acute stage of the infection; gradually they become iodine-fast. In order to differentiate between the degeneration forms of gonococci and other diplococci frequent controls of the secretion are necessary. Degenerated gonococci are infrequently found within leukocytes; they occur oftener upon epithelial cells. In most instances, however, they are lying entirely free between the formed elements.

MILL.

Spirochetal Ulceration of Tonsils—W. WINGRAVE, *Lancet*, July 24, 1915.

Spirochetal ulceration of the tonsil is characterized by: (1) a deeply excavated and sloughing ulcer on one tonsil; (2) offensively fetid breath; and (3) the presence of spirochetes and fusiform bodies in great numbers. The cases may be divided into the acute cases which last from 4 to 7 days, and into the subacute which last from 2 to 3 weeks. The spirocheta fetida, or Vincent's spirochete, is the etiological factor in the disease.

SACHS.

Vincent's Angina—J. HARPER, *Glasgow Med. Jour.*, July, 1915.

Vincent's angina may appear in what are apparently totally different forms. It is not so rare a condition as one would suppose. It may follow an acute, subacute, or chronic course. Cases of suspected diphtheria which give a negative result on bacteriological examination should be examined for the organism of Vincent's angina, and also this should be done in cases of suspected syphilis with ulceration of the throat which do not yield to treatment and in which the Wassermann reaction is negative.

SACHS.

Paratyphoid Fever—H. ROBINSON, *Lancet*, Oct. 16, 1915.

Paratyphoid fever is a much shorter and milder disease than typhoid fever. Headache and abdominal pain are the most constant symptoms. Definite rose spots in successive crops are found in 60 per cent. of the cases. The paratyphoid bacillus may not be isolated from the feces until the fever has subsided. A quantitative Widal reaction is of great diagnostic help, when frequently repeated by one who has had much experience with this reaction and when taken in conjunction with the clinical findings.

SACHS.

The Value of the Agglutination Test in Persons Inoculated with Typhoid Vaccine—E. REISS, *Münchener med. Wochenschr.*, Sept. 21, 1915.

In persons prophylactically inoculated with typhoid vaccine the agglutination test possesses no diagnostic value whatever. MILL.

"Vaccination Spleen" Tumefaction and Typhoid Diagnosis—GOLDSCHIEDER, *Deutsche med. Wochenschr.*, Oct. 7, 1915.

The tumefaction of the spleen often ensuing after preventive typhoid vaccination may cause diagnostic difficulties. Another febrile affection may be mistaken for typhoid fever and genuine typhoid spleen for "vaccination spleen." Author found that the disappearance of the vaccination spleen occurs in about 6 per cent. of the cases in the manner that after 2 months there still persists some tumefaction and that 3 months after vaccination no swelling of the spleen is detectable. It is hence necessary that in every case of fever and splenic tumor it be determined if and when a preventive typhoid vaccination has been done, and that a close observation of the patient be instituted. Palpation and percussion of the spleen have again attained greater importance since we know that the bacteriological typhoid diagnosis is often disappointing. The spleen tumor may develop within a very brief period. Soldiers with typhoid show almost always a tumor when they report themselves ill. MILL.

Typhoid without Fever—F. MEYER, *Münchener med. Wochenschr. (Feldärztliche Beilage)*, Oct. 5, 1915.

By reason of the bacteriological examination of the blood and feces we are enabled to recognize certain disease pictures as typhoid fever which are entirely discrepant from the clinical resemblance to the classical symptom-complex of this disease. We know for a long time that genuine typhoid fever may not present any intestinal symptoms. On the other hand meningitides, pneumonias and renal affections may be caused by the clinical localization of the typhoid bacillus. Hence the clinical diagnosis has to a certain degree yielded to the bacteriological examination and thus considers as typhoid all those infections which are due to the typhoid bacillus. It is here, of course, presupposed that the bacillus has really found its way into the tissues and caused disease phenomena. Accepting this definition of typhoid fever, author and others have lately approached the question whether or not there may be typhoids without fever. In other words, can a normal vigorous body be the host in his organs and the blood current of bacilli without reacting by an increase of body temperature and general or localized disease phenomena? Author describes 3 cases, taking the positive standpoint. These patients were not carriers of bacilli as was evidenced by the many fecal examinations. MILL.

Nephrotypoid—F. DEUTSCH, Wiener klin. Wochenschr., Sept. 9, 1915.

Two young girls living in the same house were affected with acute nephritis and high fever. The urine of both patients contained large numbers of typhoid bacilli. There were no other symptoms of typhoid. A brother of the girl who was first infected had died a short time before. He had also suffered from nephritis and there is little doubt that this was likewise of typhoid origin. MILL.

Number and Forms of White Cells in Typhus Fever—M. MATTHES, Münchener med. Wochenschr., Oct. 5, 1915.

The ordinary findings in typhus fever, viz: moderate leukocytosis and preponderance of polynuclear cells, in dubious cases point to typhus and not to typhoid. The blood picture of the latter is characterized by leukopenia with a synchronous lymphocytosis. The blood picture in cases of typhus fever with a low leukocyte count (20 per cent.) cannot be differentiated from that of measles (for the reason that the polynuclear cells are also increased) unless the presence of azurophile granula is of especial import. A relative polynucleosis in the presence of a low general count may indicate typhus fever. A total count of 3000 and below points to typhoid fever in dubious cases. MILL.

Artificial Hyperemia in the Diagnosis of Typhus Fever (Fleckfieber)—C. DIETSCH, Münchener med. Wochenschr., Sept. 7, 1915.

Artificial hyperemia is of value in the better recognition of a non-characteristic or unpronounced exanthema; it is of import in the differential diagnosis between typhus and typhoid; it renders the exanthema of typhus distinctly visible, and in cases, already reconvalescent or cured, it demonstrates so long as some pigmentation still persists, that the disease has been typhus. MILL.

Sero-Diagnosis of Larved Cases of Chronic Dysentery—H. STRAUSS, Deutsche med. Wochenschr., Sept. 9, 1915.

A rather large number of cases of grave colitis is due to dysentery as proved by sero-diagnosis. Sero-diagnosis may also differentiate between dysentery and paratyphoid from cases of simple, non-hemorrhagic colitis. MILL.

Demonstration of Meningococci in the Cerebrospinal Liquid—E. FRÄNKEL, Deutsche med. Wochenschr., Sept. 9, 1915.

Author mixes a few cubic centimeters of ascites-ager-bouillon with an equal amount of spinal fluid obtained by lumbar puncture. After standing in a temperature of 37 deg. C. for 12 to 14 hours, meningococci may often be demonstrated in the sediment in large numbers. MILL.

Spasmodic Symptoms in Rheumatism—F. J. POYNTON, Lancet, Oct. 9, 1915.

Poynton considers migraine, muscular spasm, paroxysmal sensory

symptoms and anginal attacks, when associated with a rheumatic history, as spasmodic symptoms in the course of this disease.

SACHS.

Pertussis—V. LANGE, *Berliner klin. Wochenschr.*, Oct. 11, 1915.

Pertussis is an infection, starting and ending with a catarrhal stage. Between the inaugural and terminating catarrh a convulsive stage ensues. This is characterized by the appearance of convulsive attacks of cough. No special catarrhal condition is responsible for the cough attacks. These are undoubtedly due to the nervous system. The irritation ensues in the central system, and it is conducted over various paths to the periphery. The therapy must be directed toward the nervous system.

MILL.

Goiter, Cretinism and Chagas' Disease—R. KRANS, F. ROSENBUSCH and C. MAGGIO, *Wiener klin. Wochenschr.*, Sept. 2, 1915.

Authors come to the conclusion that the disease-picture described by Chagas: goiter, myxedema, idiocy and diplegia, caused by *schizotrypanosoma cruzi*, is not as yet an absolutely demonstrated clinical fact.

MILL.

Actinomyces—V. Z. COPE, *Brit. Jour. Surg.*, July, 1915.

Actinomyces is frequently overlooked or wrongly diagnosed as septic or syphilitic infection, or as sarcoma. It should always be considered in the diagnosis in the case of any newly-formed sub-acute or chronic swelling in the region of the mouth, face, thorax, neck or right side of the abdomen. Infection with the fungus nearly always occurs from the alimentary tract. There are two clinical forms of the disease, the hard and the soft. The hard variety softens after two or three months. Peri-buccal infections comprise the majority of the cases. The features of the hard peri-buccal form are very characteristic and can often be diagnosed long before pathological investigation can give much help.

SACHS.

Respiratory Signs in Trichinosis—G. R. MINOT and F. M. RACKMANN, *Am. Jour. Med. Sci.*, Oct., 1915.

Authors review the histories of 102 cases of trichinosis. In 50 per cent. of these cases in which respiratory signs and symptoms were noted, 16 patients had cough without abnormal physical signs in the lungs, 17 had cough with abnormal lung signs, and 18 patients had abnormal signs in the lungs without cough. The duration of these signs seemed to depend on the length of time the temperature remained elevated, the signs disappearing as the temperature fell. If the signs in the lungs were slight, they only remained a few days, and only at the height of the fever. In 9 cases the signs were suggestive of pneumonia.

SACHS.

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